strategy. Cold-stressed endotherms may also bask in the sun to supplement endogenous heat production.

Mechanisms to Decrease Heat Loss. One way to reduce heat loss is to decrease thermal conductance (an alternative way is to reduce T_b; see heterothermy). A decrease in thermal conductance (at the same body mass) can reduce the T_{lc} and VO_2 substantially.

Body size is an important determinant of thermal conductance; larger animals have a lower mass-specific thermal conductance by virtue of their lower surface:volume ratio, although they have a higher absolute thermal conductance. Larger mammals have a lower T_{lc} than do smaller mammals, and their VO_2 is relatively less affected by low T_a (Figure 5–27). Increased body mass is not necessarily a useful response to cold stress for an individual endotherm. Increasing mass by 50% only decreases T_{lc} by about 2° C and by about 3° C for a 100% increase. However, endotherms that live in cold climates often have a higher body mass than individuals from warmer climates. This is Bergmann's rule, one of the bioclimatic laws postulated in the 19th century (see Supplement 5–3, page 190).

Thermal conductance can be minimized at low T_a by postural adjustments. A sphere has the minimum surface area:volume ratio, hence it is the energetically most effective shape. For example, mammals may curl up, and birds may retract their head and fluff up their feathers to assume a spherical shape (Figure 5–28). Many birds draw the legs into their more-or-less spherical insulation layer and tuck their bill under a wing to minimize heat loss from the uninsulated appendages.

Endotherms can further reduce their thermal conductance by using external insulation, i.e., nesting material. For example, lemmings with a nest of cotton wool can reduce their effective thermal conductance by 40%. Weasels will use lemming nests when resting and add to the insulation of the nest with lemming fur. Bank voles at a T_a of 4° C can reduce their daily energy expenditure from 3.5 kJ g^{-1} day^{-1} without nesting material to 2.3 kJ g^{-1} day^{-1} with nesting material.

A group of individuals can collectively reduce their thermal conductance by huddling together. Bank voles at 4° C can reduce their daily energy expenditure from 3.5 to 3.0 kJ g^{-1} day^{-1} by huddling with three other voles.

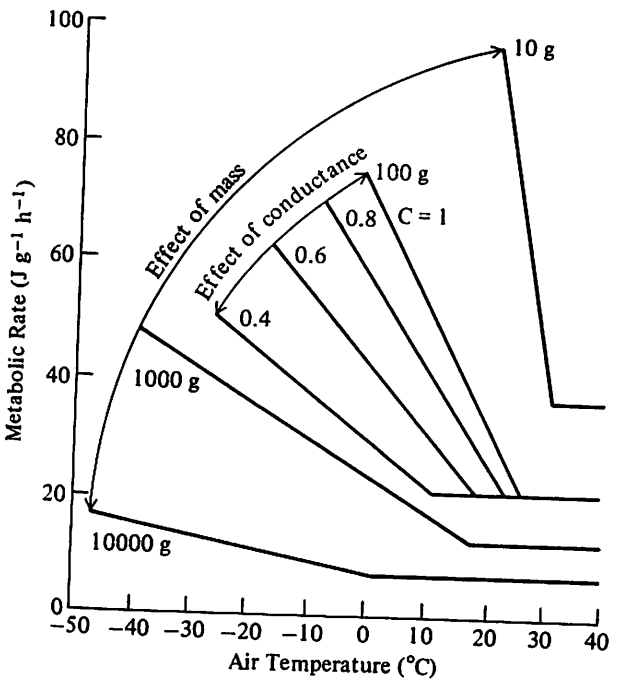


FIGURE 5–27 Predicted relationship between metabolic rate and air temperature for a 10 g, 100 g, 1000 g, and 10000 g mammal showing the decrease in basal metabolic rate, thermal conductance, and lower critical temperature (T_{lc}) with higher body mass. Shown also is the effect of decreasing thermal conductance for a 100 g mammal from 1.0 to 0.8, 0.6, and 0.4 × the normal value.

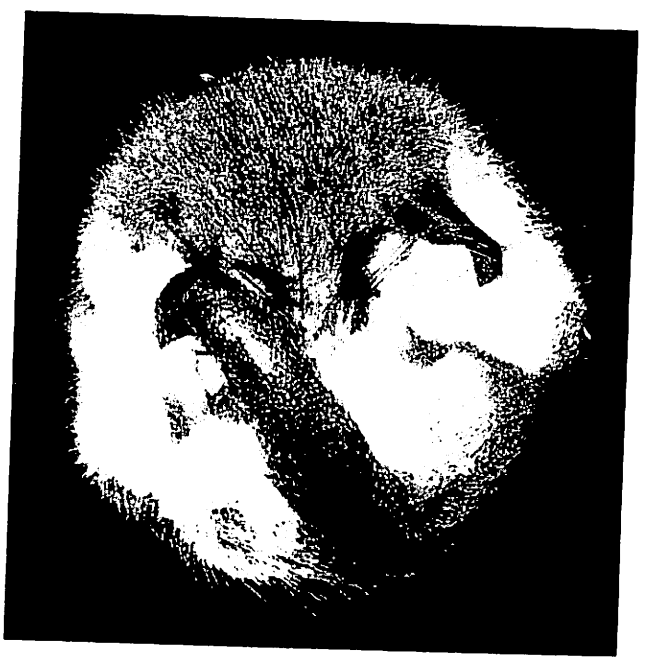


FIGURE 5–28 The typical hibernation posture of the western pygmy possum is an almost spherical ball. This minimizes the surface-to-volume ratio. *(From Geiser 1985.)*

Some mammals and birds seasonally alter their insulation to reduce conductance in winter (and at the same time change coat color to white for cryptic coloration). For example, some arctic mammals have a seasonal change in thermal conductance

$$\begin{aligned} \text{Winter: } C &= 13.9\,g^{-0.534} \\ \text{Summer: } C &= 23.5\,g^{-0.534} \end{aligned} \qquad (5.17)$$

(calculated from Casey, Withers, and Casey 1979). However, some arctic mammals such as least weasels do not have a summer–winter change in thermal conductance. These weasels also have a higher than expected conductance for a mammal because of their elongate shape and have a high metabolic rate.

A thick layer of subcutaneous fat will not only add insulation but will also decrease the surface:volume ratio with little increase in metabolic rate (since fat has a low metabolic rate). Thermal conductance can be reduced by restricting blood flow to the skin and by allowing skin temperature to decline because heat loss across the fur/feather insulation is proportional to $(T_{skin} - T_a)$ rather than $(T_b - T_a)$.

Evaporative water loss generally doesn't contribute significantly to thermal conductance at low T_a. Cutaneous evaporative water loss is low since T_{skin} is reduced and the ambient relative humidity is high. Respiratory evaporation is potentially high because of the elevated VO_2 but is minimized by a reduced expired air temperature (T_{exp}) and nasal countercurrent heat exchange (see Chapter 16). For example, the grey seal has a T_{exp} as low as 6° C (at T_a = −30° C) and can conserve up to 70% of the heat that would be lost if $T_{exp} = T_b$ (Folkow and Blix 1987). Arctic mammals similarly conserve respiratory heat loss. For example, lemmings can reduce respiratory heat loss to <5% of total heat loss at low T_a.

Mechanisms to Increase Heat Production. The principal means for increasing heat production is an increased metabolic heat production by skeletal muscle. Skeletal muscle has a high aerobic metabolic capacity and is a considerable proportion of the body mass; hence, it is capable of considerable supplementary heat production.

Skeletal muscles usually contract to move parts of the body (see Chapter 10) but it is relatively straightforward for muscle contractions to be rendered nonlocomotory. **Shivering** has been reported for many mammals (monotremes, marsupials, and placentals) and birds. In birds, there tends to be a clear inverse relationship between shivering activity (measured as electromyograph activity) and T_a, suggesting that shivering is the primary thermogenic response to cold (Figure 5–29).

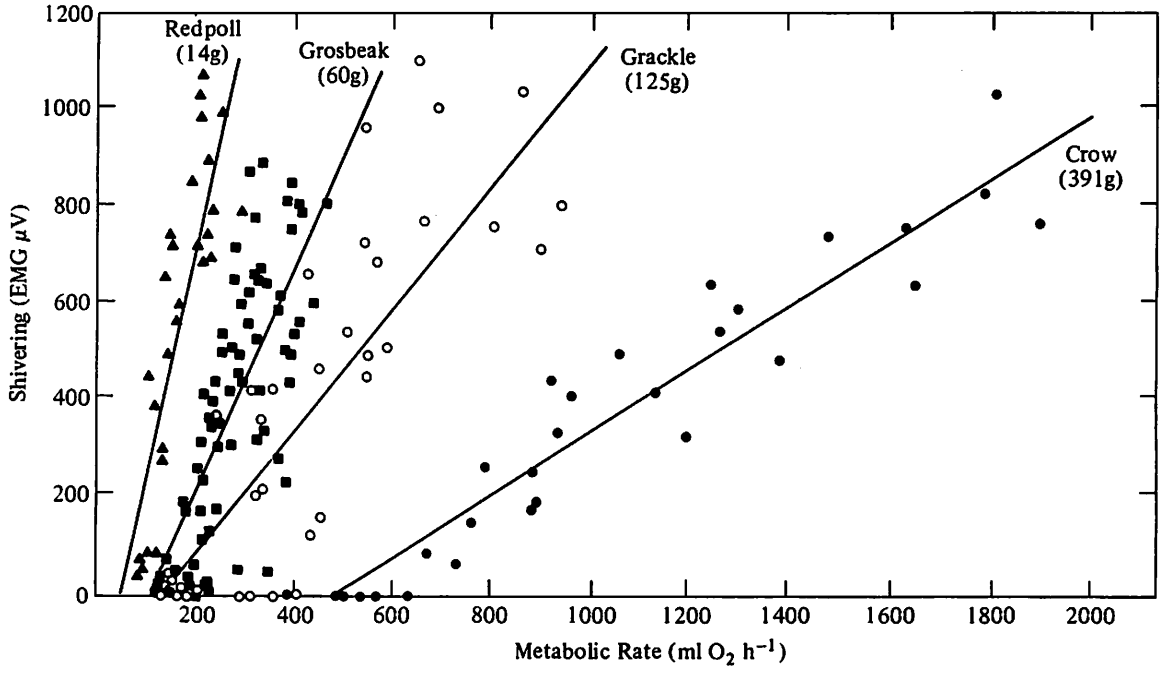


FIGURE 5–29 Linear relationship between degree of shivering (measured as electromyogram electrical activity) and metabolic rate for species of four birds. *(From West 1965.)*

Shivering is a myotactic reflex oscillation due to muscle spindle activation by γ-efferent nerve fibers to the muscle spindle (see Chapter 8). Higher brain centers (perhaps the cerebellum) are required for the oscillatory muscle contractions of shivering; shivering does not occur below the level of spinal cord transection. A few endothermic pythons also shiver and some endothermic insects "shiver" with their wings (see below). Shivering thus appears to be a fairly generalized response of many diverse endotherms to cold. It may have evolved from a generalized activity at low temperature through the acquisition of a neural capacity to repetitively stimulate muscle contraction with no gross movement (Whittow 1973).

Nonshivering thermogenesis (NST) is a second mechanism for augmented metabolic heat production. It has been reported for many placental mammals, some marsupials, and a few birds. NST is more important in small mammals, and can increase VO_2 to about 2 to 4 × basal metabolic rate (Figure 5–30).

Brown adipose tissue (BAT, or brown fat) is a type of mammalian adipose tissue that is specialized for metabolic heat production (Chapter 3). It has been identified only in placental mammals (chiropterans, insectivores, rodents, lagomorphs, artiodactyls, carnivores, and primates; Smith and Horowitz 1969). A superficially similar adipose tissue has been reported for a marsupial (Loudon, Rothwell, and Stock 1985). It has not been identified in birds. BAT is present in some hibernating mammals, some cold-adapted mammals, and some newborn mammals. It releases heat from a futile mitochondrial electron-transport cycle that produces heat without the necessity of ATP synthesis and degradation. BAT can produce remarkable amounts of heat, up to 500 J sec^{-1} kg^{-1} (cf. active skeletal muscle produces 50 to 60 J sec^{-1} kg^{-1}). Such high metabolic rates require a substantial O_2 supply; the BAT of cold-stressed rats can receive up to 1/4 of the total cardiac output (Table 5–12). Not surprisingly, BAT is an important component of nonshivering thermogenesis in many mammals, although other tissues (e.g.,

TABLE 5–12

Total oxygen consumption rate (VO_2; ml O_2 g^{-1} hr^{-1}) and oxygen delivered to brown adipose tissue (BAT) and other tissues in cold-acclimated rats at varying air temperatures. Oxygen delivery to tissues is calculated from tissue blood flow and arterial O_2 content (16.3 ml/100 ml blood) and is expressed as a percentage of the total cardiac O_2 delivery (% COD). *(Data from Foster and Frydman 1979.)*

	Air Temperature (°C)			
	−19	*−6*	*+6*	*+21*
Blood flow (% COD):				
BAT	25.0	22.6	20.2	5.6
Skeletal muscle	15.5	14.2	15.8	17.3
Heart	5.2	4.0	3.4	3.1
Kidneys	11.3	12.1	13.6	15.7
Brain	1.6	1.5	1.5	1.4
Hepatosplanchnic	14.9	12.1	15.8	19.8
Total VO_2	3.52	2.96	2.26	1.28

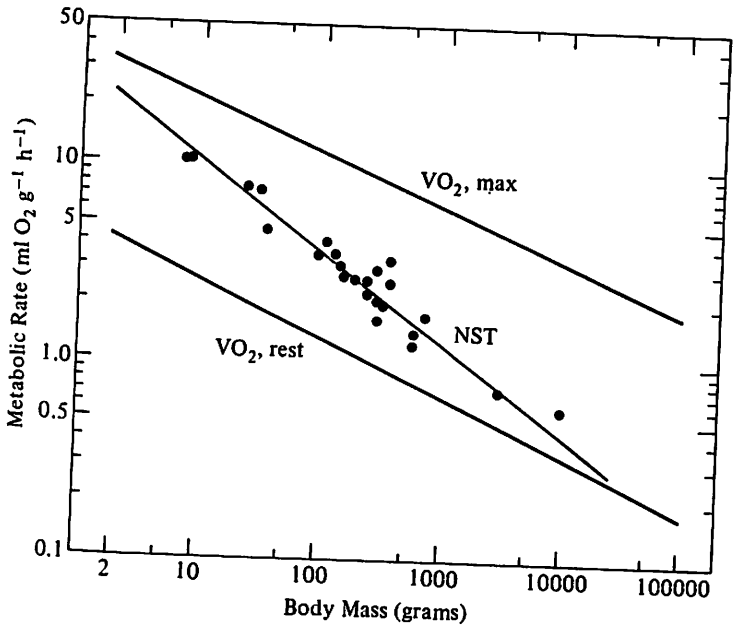


FIGURE 5–30 Nonshivering thermogenesis in rodents, bats, hedgehog, dog, and rabbit as a function of body mass; NST VO_2 (ml O_2 g^{-1} h^{-1}) = 30 $g^{-0.454}$. Upper line indicates the predicted maximal VO_2 and the lower line indicates the predicted resting VO_2. *(From Heldemaier 1971.)*

liver) can also contribute to NST at moderate T_a. The Na^+/K^+ "leak" of endotherm cells could also provide an important mechanism for NST.

Some young and adult birds appear to have NST, but not BAT. For example, king penguin chicks have a lower shivering threshold temperature (STT = −18.5° C) when cold acclimated than when maintained at 25° C (STT = −9.1° C: duChamp et al. 1989). The site for NST may be visceral organs (e.g., liver) or skeletal muscle. Some cold-acclimated birds do have a specialized fat tissue (it is highly vascular and multilocular), but this is probably a highly mobilizable fat store, rather than a thermogenic tissue.

Heat production by other biochemical processes may substitute for shivering and nonshivering thermogenesis. For example, the specific dynamic effect (SDE) of digestion can reduce the required shivering and nonshivering thermogenesis (see Chapter 4). The heat of fermentation can substitute for shivering and NST thermogenesis for ruminant and pseudoruminant mammals.

Heat produced by general activity (e.g., during the activity phase of the circadian cycle) might also be expected to contribute to thermoregulation if generalized activity was the evolutionary precursor activity for shivering. However, an increase in general activity would at the same time promote heat loss by altering the body posture (e.g., extension of appendages for locomotion); minimizing boundary layer thickness; and disrupting the fur or feather insulation layer, i.e., generalized activity would increase thermal conductance. It is significant in this respect that both mammals and birds have lower thermal conductance in their inactivity phase than in their activity phase. Activity would also prevent shivering thermogenesis (which doesn't have associated detrimental effects on thermal conductance). In practice, exercise appears to partly substitute for shivering and NST. For example, exercise can partly substitute for shivering/NST at T_a < 10° C for white rats acclimated to 30° C, but only at lower T_a (< −20° C) for rats acclimated to 6° C because of their greater NST (Jansky and Hart 1963). Exercise thermogenesis apparently only partially substitutes for shivering and NST since T_b declines at low T_a.

Finally, solar radiative heat gain can substitute for metabolic heat production. Cold-stressed endotherms can bask in sunlight to minimize their metabolic heat production requirements. For example, herring gulls have a lower VO_2 at low T_a when exposed to a radiative heat load, and their T_l is reduced from about 20° C to < −5° C (Lustick et al. 1979; see also Figure 5-3A).

Heterothermy. There is a considerable metabolic cost to endothermy at low T_a despite the above-mentioned means for reducing heat loss. Consequently, some endotherms will allow the temperature of peripheral tissues, such as skin and appendages, to decline to below core T_b; this is peripheral, or regional, heterothermy. Some mammals and birds allow the core T_b to decline; this is temporal heterothermy, or torpor.

Appendages tend to have a high surface:volume ratio and high heat loss. Cold-adapted animals tend to have reduced appendages (Allen's rule; Supplement 5–3, page 190).

Heat loss from appendages is often exacerbated by the ineffectiveness or lack of insulation, e.g., mammals may have naked or poorly furred digits and tails, and birds have naked beaks and poorly feathered legs. Many endotherms cover these "thermal windows" by postural adjustments when cold stressed, but some do not, particularly if they are

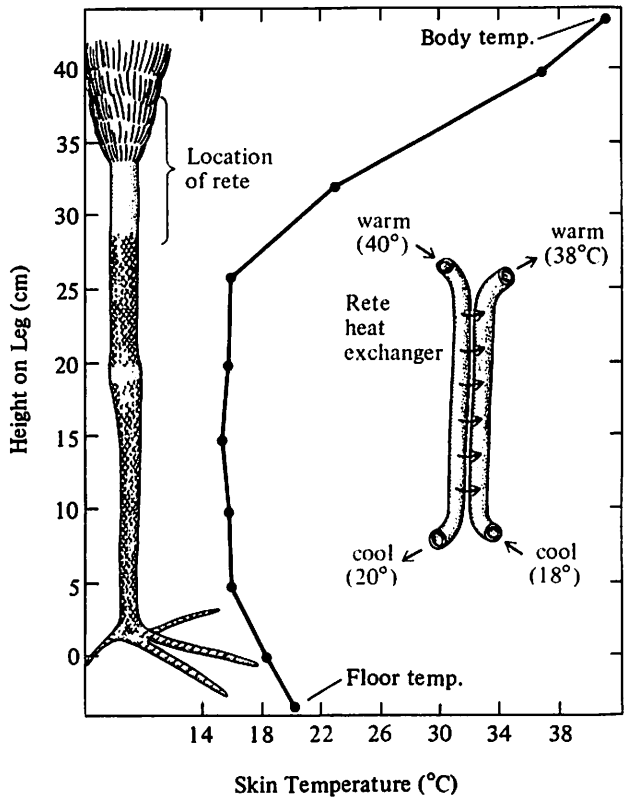


FIGURE 5–31 Skin temperature of the leg of the wood stork at an ambient temperature of 12° C and a floor temperature of 20° C. Inset shows the principle of countercurrent heat exchange in an arterial venous rete, such as that located in the stork leg. *(Modified from Kahl 1963.)*

active. For example, the glaucous-winged gull at a T_a of $-16°$ C has a core T_b of about 37.8° C but the foot skin temperature is as low as 0 to 4.9° C at the feet (Irving and Krog 1955). Similarly, the skin temperature of the wood stork's leg is close to T_a (Figure 5–31); this minimizes heat loss to the environment.

The capacity to lower the skin temperature of appendages and to minimize the loss of core body heat to hypothermic limbs is achieved by a countercurrent exchange of heat between warm arterial blood ($T_{art} = T_b$) and cold venous blood returning from the limbs ($T_a < T_{ven} < T_b$). The warm arterial blood flows in the opposite direction to the cool venous blood, and there is conductive heat transfer across the walls of the artery and veins so that the heat is lost from the arterial blood and warms the venous blood returning to the body (Figure 5–31, inset). Such a countercurrent heat exchange system has been described for limbs of a variety of mammals and birds: sloth limbs, whale fins, human arms, beaver tail, fox legs, monkey tails, and gull and stork legs.

The countercurrent exchange of heat between arterial and venous blood is facilitated by the anatomical arrangement of the blood vessels. There are three general types of countercurrent heat exchangers. In venae comitantes, a central artery is surrounded by a number of anastomosing veins, e.g., beaver hindlimbs and the forelimb of penguins (Figure 5–32A). The vascular heat exchangers in limbs and flukes of porpoises and whales have 15

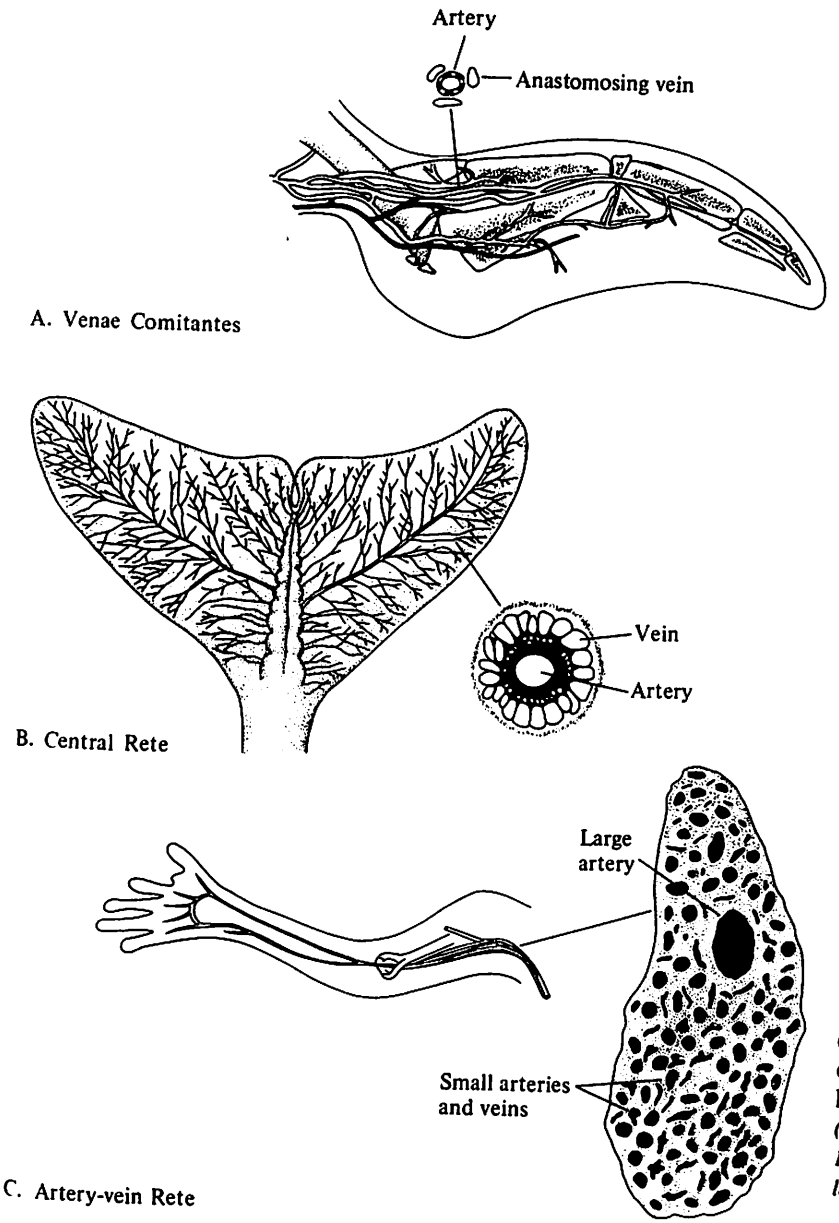


FIGURE 5–32 Three types of countercurrent heat exchangers in mammal limbs. (A) A venae comitantes in which two, three, or more anastomosing veins surround a central artery, e.g., the flipper of the jackass penguin; (B) a central artery surrounded by many small veins, e.g., the fins and flukes of cetaceans; and (C) a rete of small interdigitating arteries and veins, e.g., the limb vascular bundle of the loris. *(From Frost, Siegfried, and Greenwood 1975; Scholander and Schevill 1955; Scholander 1957.)*

to 20 small veins surrounding a central artery (Figure 5–32B). The most specialized and effective heat exchanger is a rete of small, intertwining arteries and veins, e.g., legs of wading birds, manatee limbs, beaver tail, and loris limb (Figure 5–32C).

At subfreezing air temperatures, limb countercurrent heat exchange could allow freezing of the peripheral tissues, causing frostbite and tissue death (vertebrate tissues are generally unable to survive freezing). Peripheral freezing can be prevented by conveying sufficient body heat to the limbs via arterial blood. For example, control of blood flow to the limbs enables the arctic wolf to avoid freezing of its peripheral tissues (Henshaw, Underwood, and Casey 1972). The normal countercurrent heat exchange system must be bypassed to allow warm arterial blood to reach the peripheral tissues. For example, the beaver has two countercurrent heat exchangers, a venae comitantes of arteries and veins to the hindlimbs, and a rete of small arteries and veins in the tail (Cutright and McKean 1979). Each heat exchange system has a bypass vein that returns blood to the central venous system without it passing through the countercurrent heat exchange system. These bypasses also allow heat dissipation in, for example, warm seasons or during activity.

A diverse assemblage of birds and mammals are temporal heterotherms (Table 5–13). They abandon thermoregulation at the normal T_b when cold stressed and T_b declines. In some mammals and birds, the decline in T_b may be only 4° to 8° C and T_b remains above about 30° C, but in others the decline in T_b is more profound; the T_b may decline to almost equal T_a and may be less than 5° C.

An adaptive decline in core T_b is **hypothermia.** There is a considerable, and confusing, terminology for the various patterns of hypothermia. Hypothermia may be natural or experimentally induced. The criterion for natural hypothermia is that the animal is able to spontaneously arouse (rewarm) to its normal T_b using endogenous heat production (typically shivering or NST). **Torpor** is a pronounced natural hypothermia accompanied by a substantial depression of metabolism, respiratory rate, heart rate, and lack of motor coordination and response to external stimulation. The term dormancy is also applied to natural hypothermia, but has other usages (e.g., dormancy of plant seeds, winter dormancy of amphibians and reptiles). Hibernation is also a widely used term to describe long-term torpor in response to winter cold and food deprivation. Unfortunately, it is also used to describe winter dormancy of reptiles (a different physiological phenomenon) and the mild hypothermia of bears. To avoid confusion, the term torpor is used here to describe the physiological state associated with pronounced hypothermia (T_b < 30° C) as opposed to mild hypothermia (T_b > 30° C). **Estivation** is a torpid state induced by lack of food and/or water during high environmental temperatures; it is physiologically indistinguishable from torpor, except for the higher

TABLE 5–13

Taxonomic distribution of temporal heterothermy in mammals and birds, including mild hypothermia (in parentheses) and torpor.

Birds		
Nonpasserines	Columbiformes	(inca dove)
	Cuculiformes	(ani, roadrunner)
	Strigiformes	(snowy owl)
	Falconiformes	(turkey vulture)
	Caprimulgiformes	poorwill
	Apodiformes	Apodidae; swifts
	Trochiliformes	Trochilidae; hummingbirds
	Coliformes	Mouse-birds
Passerines	Passeriformes	Hirundinidae; swallows
		(Paridae; chickadees, tits)
		(Ploceidae; sparrows, weavers)
		(Fringillidae; finches)
		(Nectarinidae; sunbirds)
		(Pipridae; manakins)
		(Icteridae; new-world blackbirds)
Mammals		
Monotremes	Tachyglossidae	echidna
Marsupials	Didelphidae	American marsupials
	Dasyuridae	marsupial mice
	Phalangeridae	possums
	Tarsipedidae	honeypossum
Placentals	Rodentia	Sciuridae; squirrels
		Muridae; murids
		Cricetidae; cricetids
		Heteromyidae; kangaroo rats, mice
		Dipodidae; jerboas
		Gliridae; dormice
		Zapodidae; jumping mice
	Primates	mouse lemurs
	Megachiroptera	fruit bats
	Microchiroptera	other bats
	Insectivora	insectivores
	Carnivora	(bears, badgers)

T_b during estivation. It will be briefly described below as an adaptation to heat stress.

A number of birds and mammals use moderate hypothermia as a short-term response to cold stress. For example, the tropical manakins *Manacus* and *Pipra* (small frugivorous passerines) have a normal T_b of about 37.9° C but starved birds will become hypothermic at night, with T_b dropping to 27° to 36° C. This hypothermia significantly reduces the metabolic rate to 58% of the normal value (Bartholomew, Vleck, and Bucher 1983). A number of other birds use short-term hypothermia to conserve energy; these include the turkey vulture, smooth-billed ani, inca dove, and the snowy owl. Some mammals also use moderate hypothermia in response to cold. For example, the marsupial mouse *Antechinus stuartii* may have a moderate depression of T_b (up to 5° C) when inactive in moderate and cold environments. Hibernating bears and some other carnivores, such as badgers, have only a moderate (5° to 10° C) decline in T_b when inactive in the cold. For example, the T_b of black bears declines to 31° to 35° C during winter dormancy; heart rate declines from 50 to 60 min^{-1} to 8 to 12 min^{-1} and VO_2 declines to 32% of normal levels. The term "carnivore lethargy" is sometimes used to distinguish this moderate hypothermia from torpor.

Deep hypothermia, or torpor, has been reported for a variety of mammal and bird families. A typical torpor cycle has three stages: **entry** into torpor, the **prolonged period** of torpor, and **arousal** from torpor (Figure 5–33). T_b declines markedly during torpor, often to within 1° to 2° C of T_a. Associated with the profound decline in T_b is a marked decline in metabolism. There is an obvious energetic savings associated with torpor.

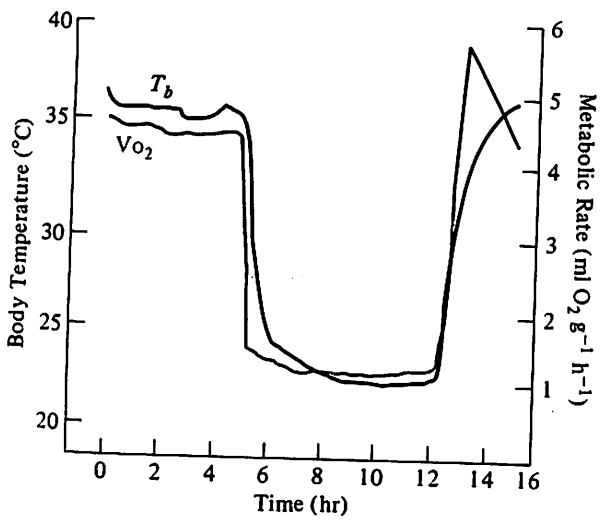


FIGURE 5–33 Metabolic rate and body temperature of a deer mouse during a typical daily torpor cycle. *(From Nestler 1990.)*

Torpor is not an abandonment of thermoregulation and assumption of ectothermy but is a controlled physiological state. This is evident from the relationship between T_b and VO_2 of torpid endotherms at low T_a. The T_b is generally close to T_a (T_b must be a degree or so above T_a to dissipate metabolic heat). However, there is a minimum **critical body temperature** ($T_{b,crit}$), which a torpid endotherm will maintain during torpor, even if T_a drops below the $T_{b,crit}$. Endogenous heat production is increased to maintain T_b at $T_{b,crit}$, and the elevation in heat production is proportional to $(T_b - T_a)$. For example, the hummingbird *Eulampis* has a $T_{b,crit}$ of about 15° C; T_b is similar to T_a when torpid at $15 < T_a < 30$° C, but the T_b is regulated at about 15° C by elevated metabolic heat production when $T_a <$ 15° C (Figure 5–34). The increase in VO_2 at $T_a <$ 15° C is parallel to that observed for euthermic hummingbirds, since the slope of this relationship is the thermal conductance (which is similar for euthermic and torpid birds). A very similar pattern of T_b regulation during torpor is observed for mammals during torpor, e.g., the shrew *Suncus* has a $T_{b,crit}$ slightly less than 15° C. Many torpid birds and mammals have lower $T_{b,crit}$; for example, the pygmy and honey possums have a $T_{b,crit}$ of about 5° C. Torpor at $T_a < T_{b,crit}$ is thus a highly regulated physiological state equivalent to normal thermoregulation, except that the setpoint for T_b regulation is lowered to $T_{b,crit}$.

Entry into torpor is generally very rapid, particularly for small birds and mammals. Some animals show a few "test-drops" of VO_2 then rapidly enter torpor. It is energetically advantageous to enter torpor as rapidly as possible because this maximizes the energy savings. Entry into torpor appears to be a fairly passive cooling response to the abandonment of normal thermoregulation. Animals do not, for example, shiver during entry into torpor to regulate the rate of cooling. Hummingbirds and the poorwill enter torpor at a rate determined by their passive cooling properties. The rate of entry into torpor could be maximized by increasing thermal conductance, for example by a postural adjustment of ptilo/pilo-depression, but there is little evidence that this occurs. Consequently, the rate of entry into torpor is proportional to the normal thermal conductance, i.e., $\propto$ mass$^{-0.5}$. Small mammals and birds (2 to 10 g) therefore enter torpor much more rapidly than

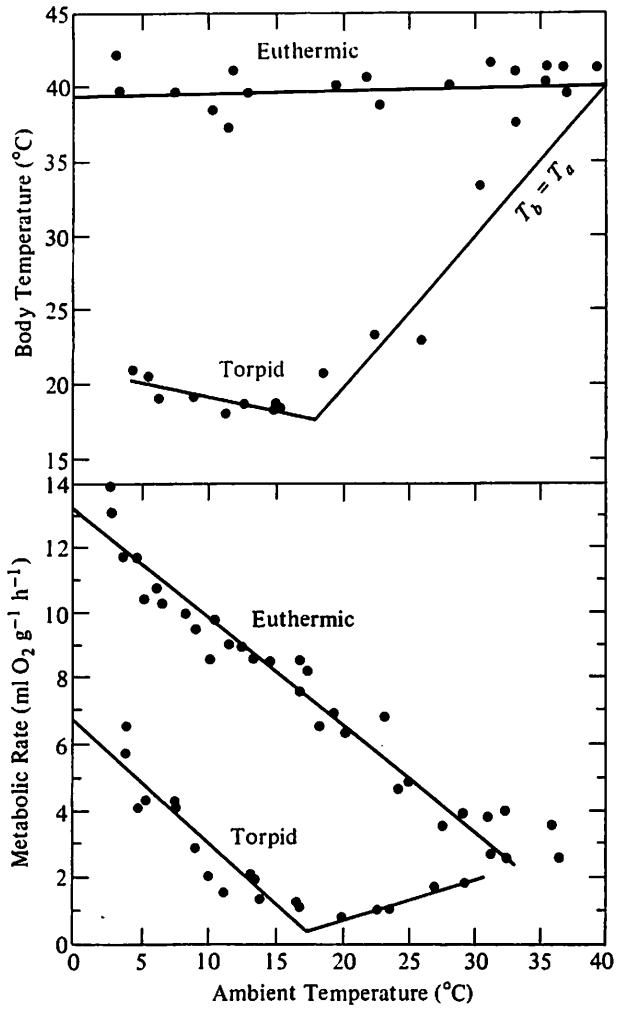


FIGURE 5–34 Relationship between metabolic rate, body temperature, and ambient temperature for euthermic and torpid hummingbirds. *(Modified from Hainsworth and Wolf 1970.)*

TABLE 5–14

Times required for entry into torpor and arousal from torpor, calculated from allometric relationships between rates of entry into torpor and arousal for a variety of birds and mammals of varying body mass. Calculations are for cooling and arousing at an ambient temperature of 15° C, with body temperature changing from 37° C to 17° C (note that some of the animals listed can only tolerate mild hypothermia).

	Body mass (grams)	Entry Time[1] (min)	Arousal Time[2] (min)
Shrew *Suncus*	2	35	13
Hummingbird *Archilochus*	4	59	17
Honey possum *Tarsipes*	10	80	24
Poorwill *Phalaenoptilus*	40	224	41
Nightjar *Eurostopodus*	86	350	55
Turkey vulture *Cathartes*[3]	230	2336 (39h)	190 (3.2h)
Echidna *Tachyglossus*	3500	1648 (27h)	226 (3.8h)
Marmot *Marmota*	4000	1766 (29h)	237 (4.0h)
Badger *Taxidea*[3]	9000	2685 (45h)	323 (5.4h)
Bear *Ursus*[3]	80000	8307 (138h)	741 (12.3h)

[1] Calculated from the thermal conductance.

[2] Calculated from arousal rate (°C min^{-1}) = 1.97 $g^{-0.38}$.

[3] Can only tolerate mild hypothermia..

larger mammals and birds. A 2 g shrew would cool from 37° to 17° C in about 35 min at a T_a of 15° C, whereas an 80 kg bear would require about 138 hr to cool by the same amount (Table 5–14).

The metabolic rate during torpor is profoundly depressed, to 1/20 to 1/100 of the normal euthermic value. The magnitude of the decrease in VO_2 reflects two factors. First, there is a marked decline in VO_2 due to the abandonment of thermoregulation at low T_a, i.e., the difference between cold-stressed VO_2 and $VO_{2,basal}$. Second, there is a subsequent decline in T_b, hence VO_2, due to a Q_{10} effect as T_b declines. The energy saving, due to a torpor cycle, is considerable, depending on body mass, T_a, and Q_{10}. The energy saving is more for a small mammal or bird because small endotherms have a higher thermal conductance and T_{lc}, and their metabolic rate is increased more by low T_a (Figure 5–35). However, the energy savings from a decline in T_b is similar for a small and large endotherm (if the Q_{10} were the same). The Q_{10} for metabolic rate depends on the body mass, whether the torpor is short or long term, and on the T_a. The Q_{10} is about 2.2 for daily torpor regardless of the T_b and body mass, but tends to be over 3 for small mammals and birds during prolonged torpor. This suggests that there are additional physiological mechanisms for depressing metabolic rate during prolonged torpor, at least for small mammals and birds.

The $T_{b,crit}$ is generally much higher than the freezing point of tissues. Is the role of such high $T_{b,crit}$ to prevent tissue freezing? Endotherm tissues

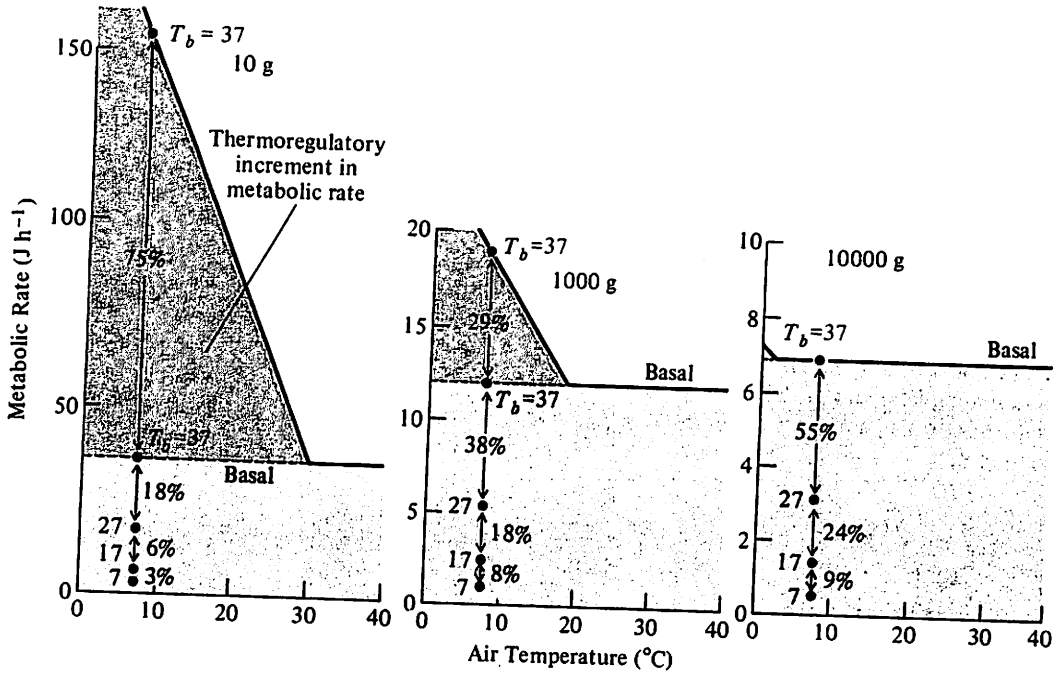


FIGURE 5–35 Predicted relationships between metabolic rate and temperature for euthermic and torpid mammals varying in mass from 10 to 10000 grams and showing the energy savings of torpor partitioned into that accruing from the abandonment of thermoregulation and that from the decline in body temperature.

can supercool to avoid freezing; for example, the torpid arctic ground squirrel can safely supercool to a core temperature of −2.9° C (Barnes 1989). This suggests that the role of $T_{b,crit}$ is not in antifreeze protection. It more likely reflects the difficulty of arousal from torpor (see below).

There are a number of different patterns of torpor, with respect to its depth and duration. Some species have a shallow torpor ($T_{b,crit}$ > 12° C), whereas others have much deeper torpor ($T_{b,crit}$ < 12° C). There is, in fact, a continuum in the $T_{b,crit}$ of various torpid endotherms but 12° C seems to be a reasonable arbitrary breakpoint to distinguish shallow from deep torpor. Many birds and mammals become torpid on a circadian cycle, and arousal occurs within 16 to 24 hours of the onset of torpor. In other birds and mammals the torpor bout can be prolonged, lasting many days, weeks, or even months. Daily torpor is often of the shallow type (e.g., dasyurid marsupials, many cricetid rodents, shrews) but may be deep (e.g., the marsupial honey possum and pygmy possum, heteromyid rodents). Long-term torpor is generally deep (e.g., pygmy possums, squirrels) but may be shallow (e.g., some insectivores, carnivores).

Torpor is terminated by spontaneous arousal. The metabolic rate increases rapidly and T_b rises to the normal levels. The torpid endotherm must generate sufficient metabolic heat to arouse, but its maximal metabolic rate depends on T_b. For example, $VO_{2,max}$ (ml g^{-1} hr^{-1}) of a pocket mouse *Perognathus* is 0.38 T_b − 2.8, and for a honey possum *Tarsipes* it is 0.45 T_b − 2.1. Consequently, there is a minimum T_b below which arousal is not possible; this would be similar to, or lower than, the $T_{b,crit}$.

The arousal rate should depend on the initial T_b and body mass, since mass-specific metabolic rate is proportional to mass$^{-0.25}$. At a T_b of 20° to 25° C the arousal rate (°C min^{-1}) of torpid birds and marsupial and placental mammals is about 1.8 $g^{-0.3}$ (Figure 5–36). Large endotherms have longer arousal times than small endotherms (Table 5–14). Very large mammals (e.g., bears) have a very long arousal time. This, and their long entry time, indicate a minimum torpor duration of about 6 days for a bear. This would save perhaps 50% of the energy that would have been expended at normal T_b. The energy savings would be 80% if T_b = T_a for the entire torpor period. Insects warm up at a rate proportional to their thoracic mass$^{0.084}$ rather than

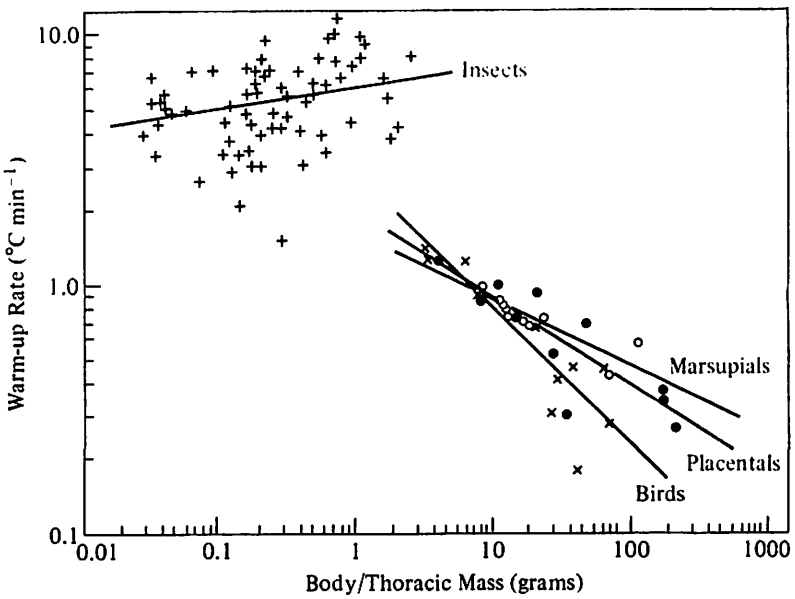


FIGURE 5–36 Rate of warming (*WR*; °C min^{-1}) for heterothermic insects during warm up and for torpid mammals (placentals and marsupials) and birds during arousal from torpor, as a function of body mass (grams).
Insects: $WR = 5.9\ g^{0.084}$
Birds: $WR = 2.7\ g^{-0.545}$
Marsupials: $WR = 1.5\ g^{-0.269}$
Placentals: $WR = 1.9\ g^{-0.351}$

being inversely proportional to mass, but their warm-up rates are similar to those predicted from the mammal/bird relationships extrapolated to the small mass of insects.

Adaptations to Heat. Most mammals and birds can readily tolerate moderate heat stress for long periods of time, but the T_a that constitutes moderate heat stress depends on their body mass. A large, well-insulated mammal would experience heat stress at a much lower air temperature (e.g., 25° C) than a small mammal or bird (e.g., >35° C). Thermal conductance is elevated at the upper end of the thermoneutral zone by nonmetabolic means such as postural change, increased peripheral blood flow, pilo or ptilo-depression, and a moderate increase in evaporative water loss. These mechanisms are no longer sufficient at $T_a > T_{uc}$ and more metabolically costly mechanisms are used, e.g., panting, gular flutter, sweating, or salivation. Body temperature may also increase to maintain a $T_b - T_a$ differential for passive heat loss (or minimize the differential for passive heat gain), but this also increases the metabolic heat production through a Q_{10} effect.

Mammals and birds are able to tolerate very high T_a (40° to 60° C) at least for short periods of time. Conduction, convection, and evaporation are avenues for heat dissipation only if $T_b > T_a$, but evaporation is the only mechanism for heat dissipation if $T_b < T_a$. Heat storage by a temporary increase in T_b (**hyperthermia**) is also an effective, though nonsteady-state mechanism employed by many mammals and birds when heat stressed.

Evaporative Heat Loss. The evaporative heat loss of endotherms increases exponentially with T_a, and evaporative heat loss can exceed metabolic heat production at a T_a of about 40° C for many birds (Figure 5–37). For birds, the maximum *EWL* (mg min^{-1}) and the corresponding maximum evaporative heat loss (J min^{-1}) are as follows (Calder and King 1974).

$$EWL_{max} = 1.03\ g^{0.80}$$
$$EHL_{max} = 2.47\ g^{0.80} \qquad (5.18)$$

The cutaneous evaporative water loss (CEWL) of birds and mammals is generally low because of the high resistance of their keratinized epidermis to water loss (see Chapter 16). Many mammals (but not rodents or birds) can markedly increase their CEWL by **sweating**. For example, humans can increase their CEWL from about 100 mg m^{-2} min^{-1} to about 23000 mg m^{-2} min^{-1} by sweating. Many other mammals also substantially increase their CEWL by sweating. Even some birds can moderately elevate their CEWL, presumably by increasing skin temperature and cutaneous blood flow.

Respiratory evaporative water loss (REWL) can be a major avenue for evaporative water loss, especially for nonsweating mammals and birds. Mammals can elevate REWL by panting (typically at a high, resonant frequency). Birds pant or **gular flutter**. Gular flutter is the movement of the moist gular (throat) region by the hyoid apparatus; it can occur in synchrony with panting (e.g., pigeons, ducks, geese, chickens) or can be independent of panting (e.g., cormorants, pelicans). Respiratory

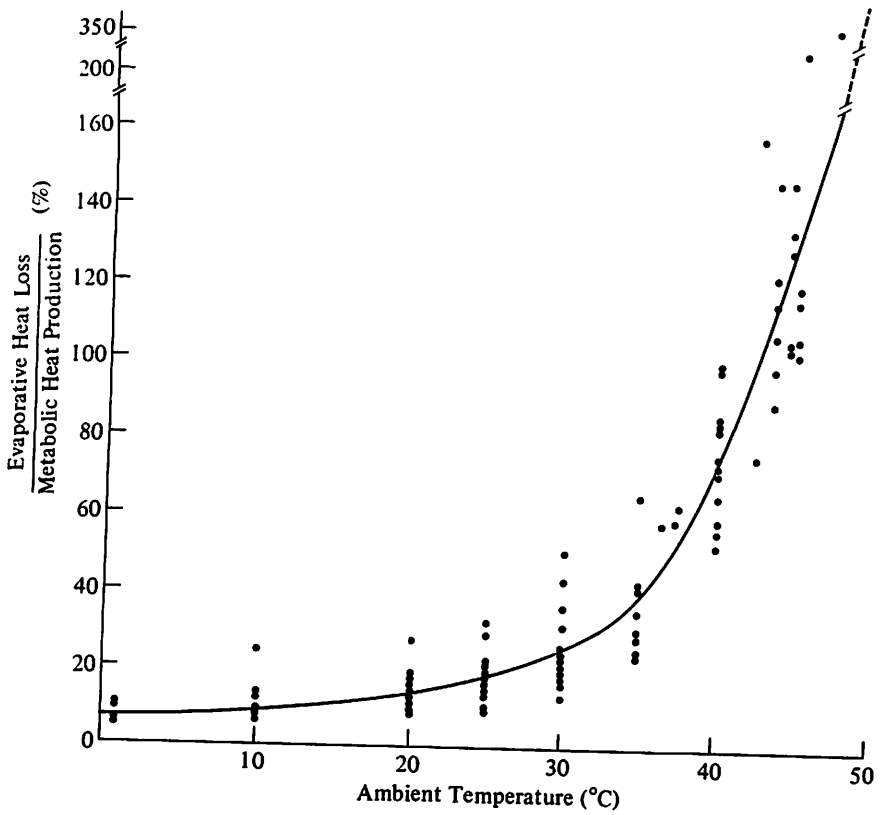


FIGURE 5–37 Evaporative heat loss per metabolic heat production at varying ambient temperatures for birds. *(From Calder and King 1974.)*

evaporative heat loss can be increased by reducing nasal countercurrent water and heat exchange (see above and Chapter 16). For example, dogs inspire through the nose and expire through the mouth when shallow panting. Many panting/gular fluttering birds do so with the mouth open. Breathing through the mouth rather than the nose also decreases the resistance to air flow.

Panting often occurs at a considerably higher frequency than resting respiration, with a rapid change from resting to panting frequency. For example, dogs abruptly increase respiratory frequency from 32 at rest to 320 min^{-1} when panting (f_{pant}/f_{rest} = 10). The panting frequency is similar to the resonant frequency of the lungs (317 min^{-1}). The pigeon and ostrich show a similar abrupt increase in respiratory frequency from rest to panting (29 to 612 min^{-1} and 4 to 40 min^{-1}, respectively). The pigeon's panting frequency is similar to the resonant frequency of its lungs (564 min^{-1}).

An increased respiratory ventilation during panting could disturb acid-base balance, causing a respiratory alkalosis (decreased blood pCO_2 and elevated pH; see Chapter 15). Some mammals and birds do experience a respiratory alkalosis, but there is little change in acid-base balance for many (see Figure 15–33, page 163). Respiratory alkalosis can be minimized, or avoided, if the increased ventilation is limited to the respiratory dead space rather than the respiratory exchange surface (alveoli in mammals, parabronchi in birds). For example, greater flamingos reduce their tidal volume when panting to 15% of the normal value, and their acid-base balance is not disturbed despite the 23 × increase in respiratory rate and 3 × increase in ventilation.

There are other possible sources of water for evaporative cooling. Some mammals (e.g., rodents and marsupials) salivate profusely when heat stressed. Some birds (e.g., the wood stork, turkey vulture, black vulture) urinate on their legs (urohidrosis) when heat stressed.

Heat Storage. Virtually all mammals and birds become hyperthermic at high T_a. This maintains a $T_b - T_a$ gradient for passive heat loss at high T_a, and minimizes the gradient for heat gain when $T_b < T_a$, but it adds to the metabolic heat load.

Hyperthermia can confer an important non-steady-state thermal advantage. Its potential significance to heat balance can be estimated as follows. An increase in T_b of 1° C absorbs about 3.5 J

g^{-1}. This is equivalent to a fractional dissipation of the hourly metabolic heat production (h^{-1}) of the following.

$$\begin{aligned} \text{Mammals: } & 0.055\ g^{0.24} \\ \text{Nonpasserine birds: } & 0.038\ g^{0.28} \\ \text{Passerine birds: } & 0.023\ g^{0.28} \end{aligned} \qquad (5.19)$$

Thus, a mammal can store more of its metabolic heat production by hyperthermia than a nonpasserine bird, which can store more of its metabolic heat production than a passerine bird. A small mammal (or bird) stores relatively less of its metabolic heat production by a 1° C hyperthermia than would a large mammal (or bird). A 10 g mammal can store 0.10 of its MHP by a 1° C hyperthermia over one hour, compared to 0.50 for a 10 kg mammal and 1.28 for a 500 kg mammal. Consequently, small mammals and birds use hyperthermia only for short-term tolerance of high T_a but larger animals can use hyperthermia for longer-term tolerance. For example, the antelope ground squirrel (100 g) is hyperthermic, with T_b up to 43° C for short periods then it returns to its burrow to cool; this cyclic hyperthermia can be repeated a number of times during the day (Figure 5–38A). In contrast, the camel (260 kg) takes a day to heat from a nighttime T_b of <35° C to a late afternoon T_b of >40° C. This daily hyperthermia cycle of the camel confers a number of advantages. First, it reduces the amount of water that must be evaporated to prevent, or minimize, changes in T_b. Second, a higher T_b facilitates heat loss to the environment and thereby minimizes the environmental heat load. Camels dehydrated by water deprivation have a more pronounced daily cycle in T_b compared to hydrated camels. This results in (1) a greater amount of heat stored in the body tissues during the day, (2) decreased evaporative water loss, and (3) reduced heat gain from the environment (Figure 5–38B).

Heat storage by hyperthermia is also important during exercise when metabolic heat production can exceed the capacity for heat dissipation. For example, gazelles running at 6 km h^{-1} store 8% of their metabolic heat by hyperthermia, but at 20 km h^{-1} the heat storage rises to 77% of metabolic heat production.

Brain Temperature Regulation. Hyperthermia may be an effective strategy for tolerating high temperatures and minimizing evaporative water loss, but it sometimes results in very high T_b's (>42° C) that could compromise the function of some body tissues. For example, the human brain tolerates temperatures up to about 40.5° C, but human core temperature may exceed 42° C (e.g., during a marathon run). Gazelles and some other mammals experience even higher T_b values.

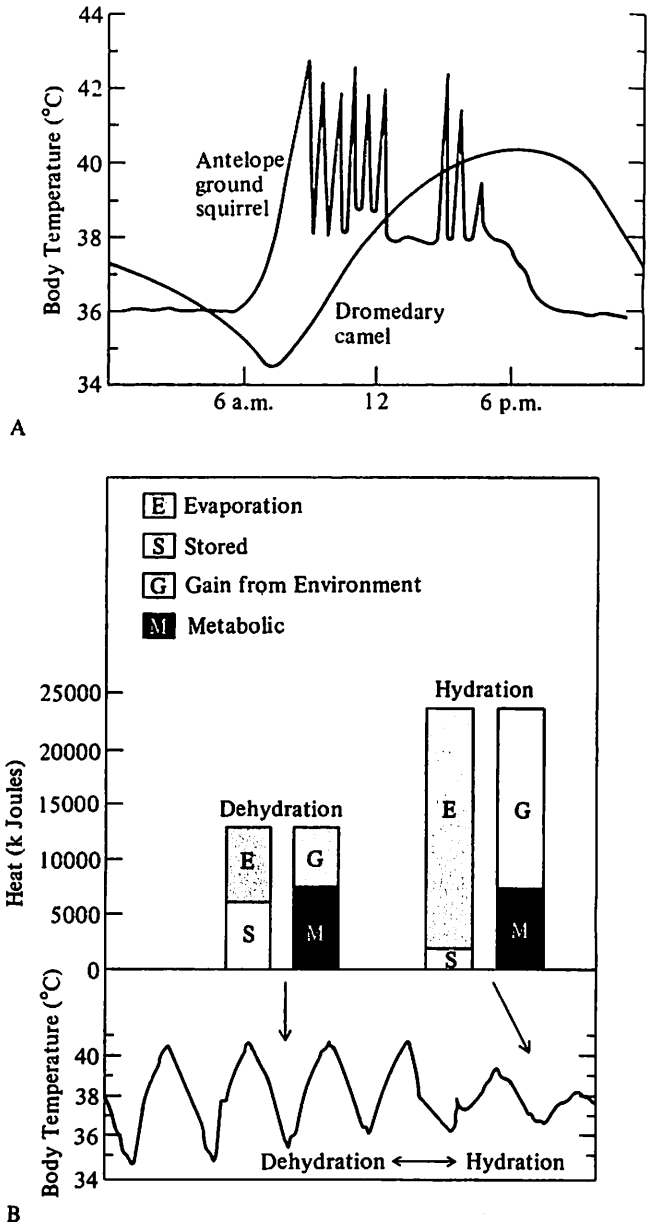
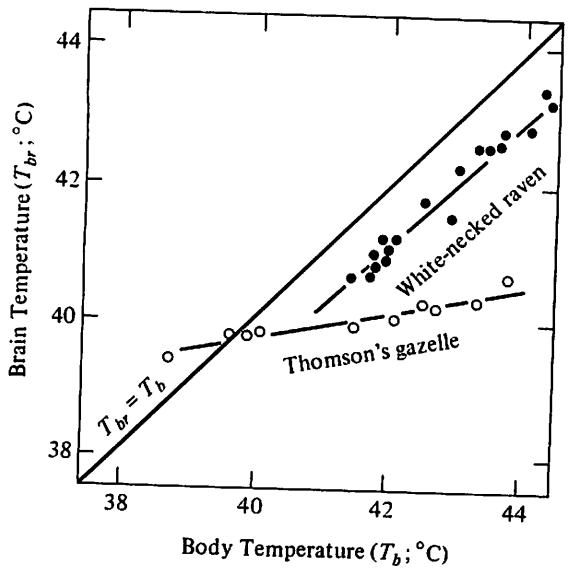
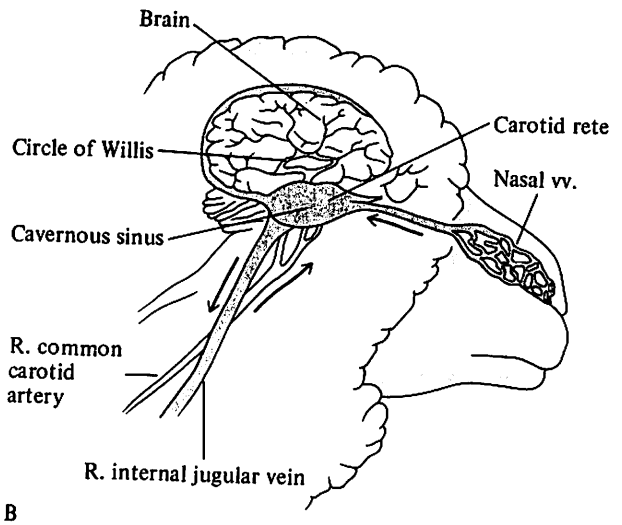


FIGURE 5–38 (A) Schematic representation of the use of short-term hyperthermia by the antelope ground squirrel *Ammospermophilus* and daily hyperthermia by the dromedary camel *Camelus*. **(B)** Effect of dehydration and hydration on daily fluctuations in body temperature of the dromedary camel *Camelus*, and a heat budget showing the magnitude of heat stored by hyperthermia, dissipated by evaporation, heat gained from the environment, and metabolic heat production. *(From Bartholomew 1964; Schmidt-Nielsen et al. 1957.)*

Many mammals and birds precisely regulate their brain temperature (T_{br}) at a lower value than body



A



B

FIGURE 5–39 (A) Brain temperature of the white-necked raven and Thomson's gazelle in relation to body temperature. **(B)** Carotid rete in the brain of a sheep allows countercurrent heat exchange between cool blood draining from the nasal mucosa and warm arterial blood passing to the brain. *(From Kilgore, Bernstein, and Hudson 1976; Taylor and Lyman 1972; Cabanac 1986.)*

temperature during hyperthermia (Figure 5–39A). This capacity is not limited to mammals and birds, but is also observed in reptiles and ectothermic flying insects. The mechanism for T_{br} regulation by mammals and birds involves evaporative cooling of blood in the nasal mucosa and subsequent heat exchange between the cool venous blood draining the nasal mucosa and warm arterial blood passing to the brain. In many mammals, the internal carotid artery forms a carotid rete of small arteries where it passes a large venous sinus, the sinus cavernosus (Figure 5–39B). Humans lack a carotid rete and do not pant when heat stressed. Nevertheless, there is probably a significant cooling of venous blood draining from the face (by sweating), and countercurrent heat exchange may occur between cool venous blood at the sinus cavernosus and internal jugular vein and warm arterial blood in the internal carotid artery. Countercurrent heat exchange in the bird brain occurs in the opthalmic rete, a network of small arteries and veins near the eye. The opthalmic rete may also exchange oxygen and carbon dioxide between the venous blood draining the nasal mucosa and arterial blood traveling to the brain (Bernstein, Duman, and Pinshow 1984).

Estivation. A variety of small fossorial mammals **estivate** during hot, dry periods. They are able to minimize thermal stress and evaporative water loss by withdrawing to the relatively cool and humid microclimate of their burrows, but prolonged periods of inactivity result in both food and water deprivation. In some species, this stress is avoided by shallow torpor cycles that occur at relatively high burrow temperatures (20° to 30° C). This shallow, summertime torpor, called estivation, is physiologically similar to winter torpor.

The cactus mouse *Peromyscus eremicus* becomes torpid during winter in response to cold stress and food restriction, and during the summer it estivates in response to either food or water restriction or negative water balance (MacMillen 1965). Estivation by the cactus mouse is characterized by relatively high burrow temperatures (about 20° C) and a high minimal T_a from which they can arouse (about 16° C). Other rodents such as ground squirrels (*Citellus*) and kangaroo mice (*Microdipodops*) estivate during the summer; kangaroo mice will estivate at a T_a as high as 28° C and as low as 5° C.

Ontogeny of Thermoregulation. Newborn and young mammals and birds generally have a poorer thermoregulatory capacity than adults. This is in part due to their smaller size, hence higher surface:volume ratio. Clearly, the young of smaller species will have more difficulty thermoregulating than the young of larger species. The generally poor thermoregulatory capacity of newborn and young mammals and birds is bolstered by a number of behavioral responses of the young (huddling) and the adults (nest building, brooding, shading).

Precocial young are relatively large at birth, have a well-developed insulation (fur or down feathers),

can move easily, and rapidly thermoregulate at adult capacity. For example, Western gull chicks (*Larus occidentalis*) are fully covered by down feathers and can move about and behaviorally thermoregulate (e.g., seek shade) within minutes of hatching. Newborn guinea pigs (that weigh 60 to 100 g) have a good fur coat, their eyes are open, and they can readily move about; they can also effectively thermoregulate over a wide range of T_a.

Altricial young tend to be small, naked, and totally dependent on their parents for survival. They are generally unable to thermoregulate by physiological or behavioral means, and rely on their parents for elevation and regulation of T_b. The young of small mammals and birds (adults < 20 g) inevitably are altricial because their very small size would confer a prohibitively high metabolic rate if they were endothermic. For example, many rodents and passerine birds have altricial young.

The development (ontogeny) of thermoregulation by altricial young is of particular interest. For example, the chicks of the masked booby *Sula dactylatra* are naked and essentially ectothermic, but their T_b is regulated at about 38° C by brooding and other behavior of the parents. They essentially have no capacity to thermoregulate until their mass exceeds about 200 g. The vesper sparrow *Pooecetes gramineus* has much smaller young (about 2 g) that are also altricial. For the first four days they are ectothermic but rapidly develop endothermy by about seven days. The small dasyurid marsupial *Dasyuroides byrnei* has extremely altricial young, as do all marsupials. The young remain in the mother's pouch for about 30 days. They are essentially ectothermic until 55 days, when they are left in the nest. Endothermy develops slowly over the next 30 or so days; the young are not fully endothermic until about 90 days old. Many placental mammals also have altricial young. The rabbit is 50 to 70 g at birth and has a sparse covering of fur. By ten days, it is better insulated and weighs 200 g and is a better endotherm than when newborn. This is primarily because of its better insulation rather than its endogenous metabolic capacity (which is actually lower per gram than that of the newborn).

There are relative advantages and disadvantages to both precocial and altricial development. The altricial strategy allows a shorter gestation period and smaller birth size for mammals, and smaller egg size for birds. Altricial species consequently tend to have larger litter/clutch sizes. The maintenance metabolism of altricial young is low because they are essentially ectothermic, so more of the ingested energy is channeled into production (growth). For example, vesper sparrow young grow at 40% mass day^{-1} while "ectothermic" (first four days). The parents provide the energy for thermoregulation, at little additional cost to their energy budget.

Reptiles

We might expect some reptiles to be endothermic because birds and mammals evolved independently from reptiles, so endothermy could well have also evolved in other reptilian groups. Furthermore, there must have been an evolutionary continuum from ectothermic reptiles to endothermic mammals and birds, so we might expect there to be some extant reptiles derived from either of these transitional endothermic lineages. However, there are few bona fide endothermic reptiles. The female Indian python *Python molurus* can regulate its T_b at about 5° to 7° C above T_a (Figure 5–40) when brooding its clutch of eggs; the T_b is elevated above T_a by shivering thermogenesis (Hutchison et al. 1966; van Mierop and Barnard 1978). The brooding diamond python *P. spilotes* is able to maintain a high T_b of about 31° C when brooding (Slip and Shine 1988).

There is not a hard-and-fast criterion distinguishing endothermy from ectothermy, and this is readily apparent for large reptiles. The extent to which reptiles can elevate T_b above T_a depends on two factors: their rate of endogenous heat production and their thermal conductance. For resting reptiles, metabolic heat production is 1.5 $g^{0.8}$ J hr^{-1} at T_b = 20° C (see Chapter 4). Thus, heat production increases with large size. The thermal conductance of large reptiles is 2603 $g^{0.148}$ J g^{-1} hr^{-1} $°C^{-1}$ for large reptiles (> 10 kg). We can approximately calculate $(T_b - T_a)$ as VO_2/C, or

$$T_b - T_a = 0.00058\, g^{0.652} \qquad (5.20)$$

This is a minimum estimate of $T_b - T_a$ because the metabolic rate can be elevated above resting by, for example, activity. How does the calculated $(T_b - T_a)$ compare with actual observations of T_b in reptiles? There is a clear trend for $(T_b - T_a)$ to be higher in large reptiles, even aquatic ones (Table 5–15).

Dinosaurs, the largest reptiles, are estimated to have weighed up to 6000 kg. There is no doubt that large reptiles (e.g., weighing over 100 kg) would be homeothermic because of their massive thermal inertia (Spotila et al. 1973), but there is considerable conjecture and debate concerning whether any dinosaurs were endothermic in the sense that mammals and birds are endotherms. That is, did any dinosaurs

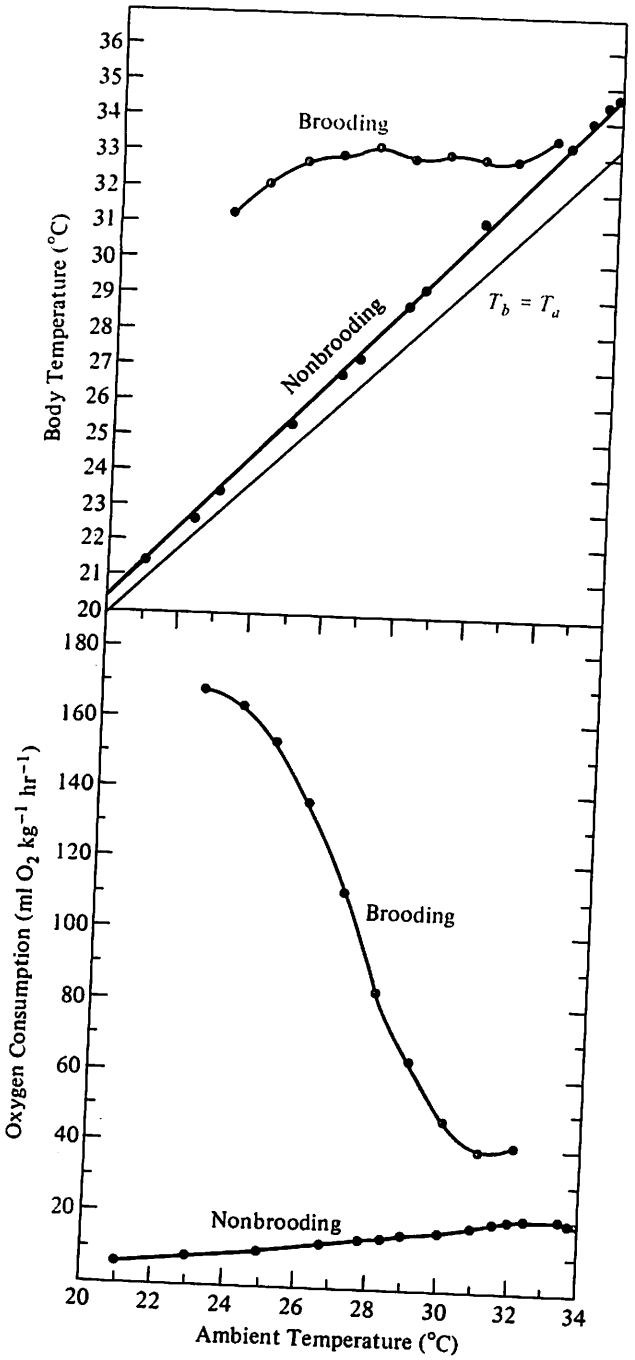


FIGURE 5–40 Relationship for body temperature and metabolic rate with ambient temperature for the nonbrooding and brooding female python. *(Modified from Van Mierop and Barnard 1978.)*

have high a resting VO_2 and use endogenous heat production to precisely regulate a high T_b? Whether any dinosaurs were endothermic is not entirely a subject of speculation, although anatomical and

TABLE 5–15

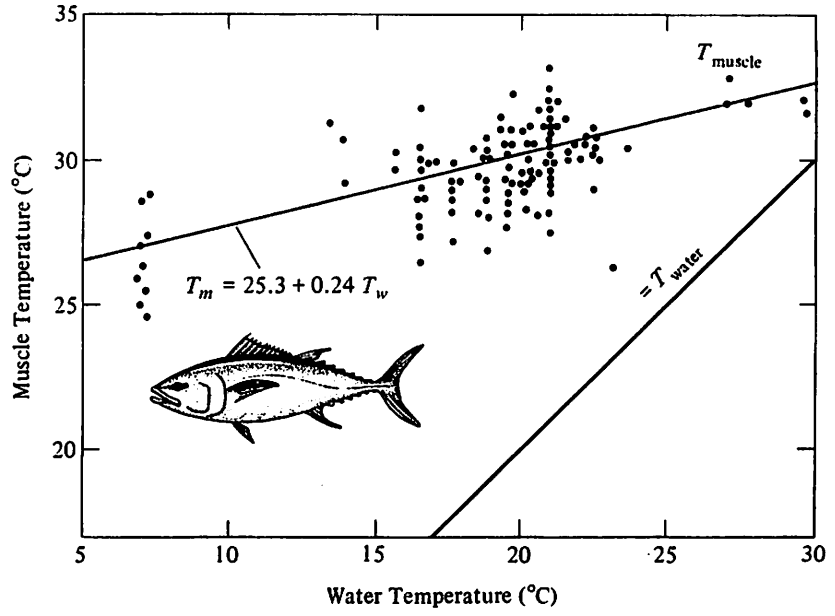
Differential between body temperature (T_b) and ambient temperature (T_a) observed for various large reptiles, and the predicted $T_b - T_a$ differential for resting reptiles in air at 20° C; for aquatic animals the predicted $T_b - T_a$ is less than, or similar to, the predicted value, depending on the body mass. Dinosaurs are shown in **bold.**

	Mass (kg)	T_a (°C)	T_b–T_a (°C)	Predicted T_b–T_a (°C)
Monitor lizard	7	25	0.2–0.4	0.2
Monitor lizard	12	25	0.2–0.5	0.3
Moschorhinid	20			0.4
Monitor lizard	35	25	0.2–0.6	0.5
Pristerignathid	50			0.7
Hawksbill/ Ridley turtles	120	28	1–3	1.2
Green turtle	127	20–30	3	1.2
Dimetrodon	150			1.4
Galapagos tortoise	170	20–30	4.1	1.5
Leatherback turtle	417	7.5	3–18	2.6
Tyrannosaur	2000			7.4
Allosaur	3000			9.6
Ceratopsid	4300			12.2
Hadrosaur	5600			14.5

physiological evidence is difficult to glean from the scanty fossil record. Advocates for the theory that dinosaurs were endothermic argue that some had an erect gait, had a Haversian bone histology, had predator-prey ratios typical of carnivorous mammals rather than carnivorous ectotherms, had a large brain, and lacked a pineal eye (as do most mammals and birds; Bakker 1971, 1972; Benton 1979). The numerous antagonists of the endothermic dinosaur theory have refuted, or at least brought into serious doubt, the validity of most of the evidence in favor of endothermy, but they generally concede that these large reptiles were at least homeothermic (McGowan 1979; Thomas and Olson 1980).

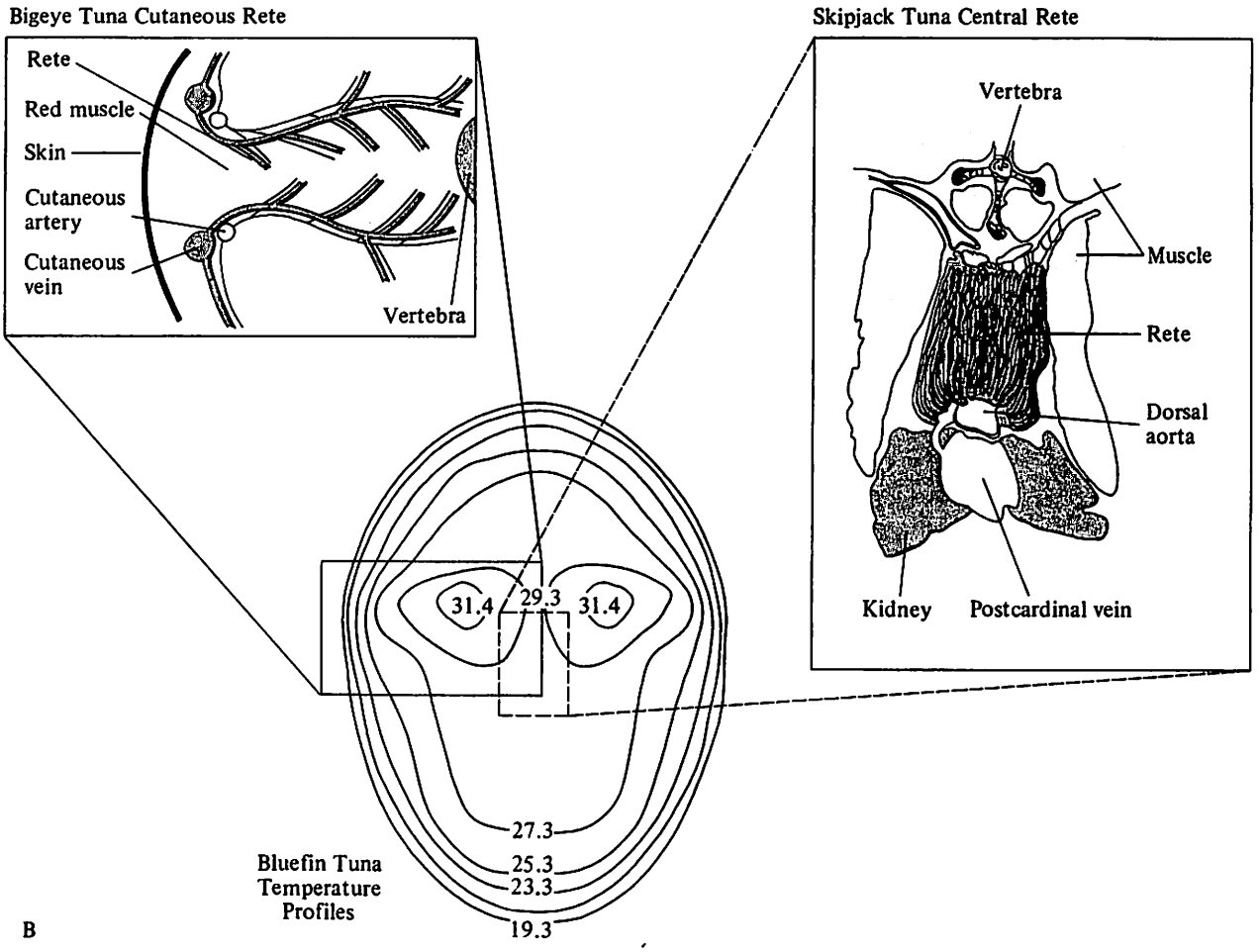
Fish

Some large and active fish can produce and retain sufficient metabolic heat to elevate tissue temperatures considerably above T_{water}. For example, bluefin tuna are large, actively swimming fish with a high metabolic rate; their muscle temperature can be 10°



A

FIGURE 5–41 (A) Relationship between muscle temperature and water temperature for bluefin tuna (*Thunnus thynnus*). (B) Temperature distribution in a bluefin tuna and arrangement of the cutaneous (bigeyed tuna) and central (skipjack tuna) countercurrent heat exchange retia. *(Modified from Stevens and Neill 1978; Carey et al. 1971; Stevens, Lam, and Kendall 1974; Carey and Teal 1966.)*



B

C or more above T_{water} (Figure 5–41A). Other tuna are also able to regulate T_{muscle} a considerable amount above T_{water}. The visceral temperature (e.g., liver) of some tuna is also elevated above T_a as are the brain and eyes (although they are not as warm as muscle). The ($T_b - T_{water}$) is highest at low T_{water}, suggesting regulation of heat retention in the muscle.

Tuna do not regulate their metabolic heat production to regulate T_{muscle} because the metabolic cost of swimming is not temperature dependent, and so their metabolic rate is essentially independent of T_{water}. This is in contrast to endothermic mammals and birds that regulate their metabolic rate to maintain T_b constant. Rather, tuna retain metabolic heat in their swimming muscle by countercurrent heat exchange in a variety of circulatory retia (Figure 5–41B). The very large tuna (e.g., bluefin) tend to have cutaneous retia, whereas smaller tuna (e.g., yellowfin) tend to have central retia (Stevens and Neill 1978). The cutaneous retia consist of a cutaneous artery and vein; the arterioles form a dense and continuous sheet that enters the muscle mass and interdigitates with a network of venules draining blood from the muscle to the cutaneous vein. The brain, eye, liver, and gut vasculature often have complex retia for countercurrent heat exchange and retention of heat in the viscera. The central retia of the smaller tuna is located beneath the vertebral column. Cool arterial blood from the gills passes from the dorsal aorta through a rete of small arteries then to segmental arteries; warm venous blood from the swimming muscle drains into segmental veins and then passes through the venous rete vessels to exchange heat with the arterial blood before entering the postcardinal vein.

A variety of other fish are also spatial endotherms, i.e., they maintain a high temperature in specific tissues. The mako, great white, and porbeagle sharks have a high visceral temperature, e.g., the stomach temperature of a mako shark may be up to 8° C warmer than T_{water}. These lamnid sharks have a peculiar routing of arterial blood through a paired vascular rete anterior to the liver. An enlarged pericardial artery forms a rete of small arteries in the lumen of a large venous space. The swordfish has a countercurrent heat exchange rete to regulate brain temperature about 4.7° C warmer and eyes 3.4° C warmer than T_{water} (Figure 5–42). There is also a mass of brown tissue associated with one of the extrinsic eye muscles; this tissue has a high density of mitochondria and cytochromes (hence the brown color) and appears to function in thermogenesis, i.e., it is similar in function and structure to brown fat of mammals.

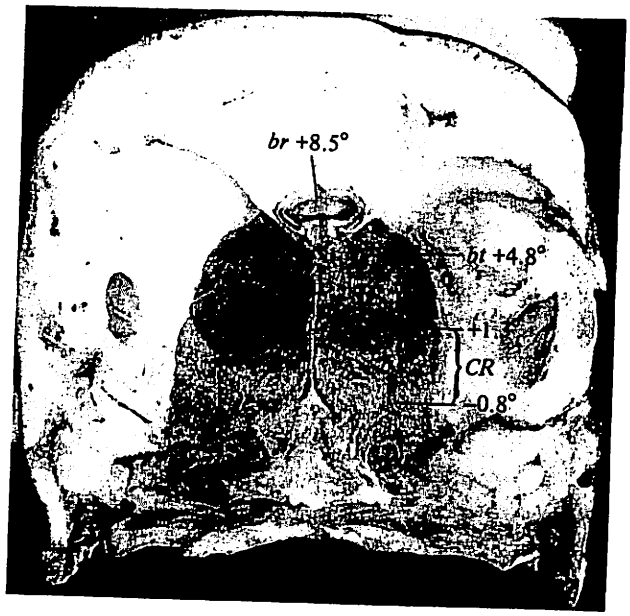


FIGURE 5–42 The brain of a swordfish (*br*) is surrounded ventrally by brown thermogenic tissue associated with the extrinsic eye muscles (*bt*). A carotid rete (*CR*) retains metabolic heat in the brain tissue for brain temperature regulation. The approximate temperatures in different parts of the head are indicated. *(Courtesy of Dr. Frank Carey, Woods Hole Oceanographic Institute 1982.)*

Insects

Many insects are endothermic and capable of precise regulation of body temperature over a wide range of T_a. Most of these are spatial endotherms that regulate a constant thoracic temperature (T_{th}) but not abdominal temperature (T_{ab}). Thermoregulation by flying insects is accomplished by the regulation of heat loss from the thorax, rather than by the regulation of metabolic heat production. This is because the metabolic cost of flight is essentially independent of T_a. Thus, endothermic insects regulate heat loss not heat production (like endothermic fish).

Perhaps the most striking example of an endothermic insect is winter-flying moths, which are active at about 0° C (Heinrich 1987). The small noctuid moth *Eupsilia* has a high T_{th} of about 30° C when flying at subzero T_a (Figure 5–43A). This is remarkable because it only weighs 100 to 200 mg. The capacity to thermoregulate at low T_a is a consequence of its effective thermal insulation on the thorax and head, and the thermal isolation of the

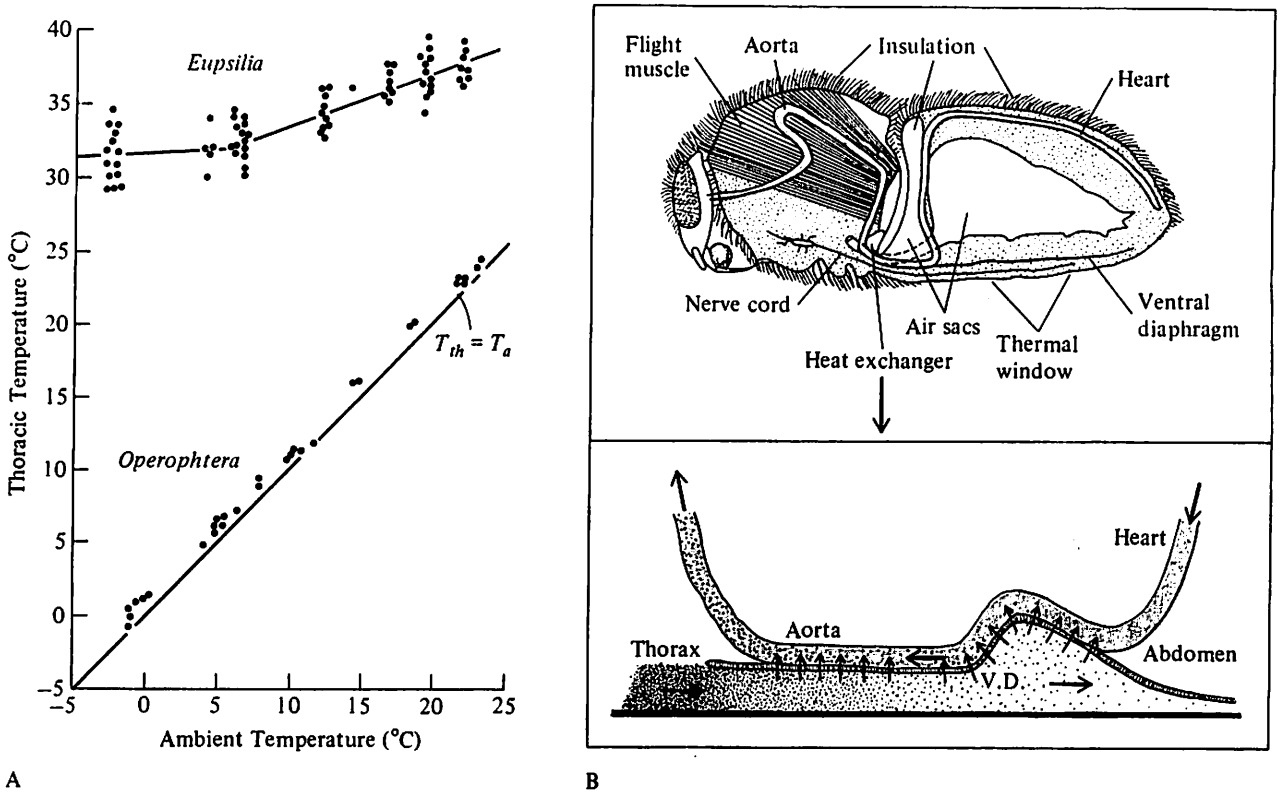


FIGURE 5–43 (A) Relationship between thoracic temperature and air temperature for two winter-flying moths, the noctuid *Eupsilia* and the geometrid *Operophtera*. **(B)** Diagrammatic cross section of a bumblebee (*Bombus vosnesenskii*) showing the thermal isolation of the thorax from the abdomen by air sacs, and the countercurrent heat exchange between blood flowing to the thorax and returning to the abdomen. *(From Heinrich and Mommsen 1985; Heinrich 1987; Heinrich 1976.)*

thorax from the abdomen by air sacs. The only significant connections between the thorax and abdomen are the esophagus, ventral nerve cord, and ventral aorta. There is countercurrent heat exchange between cool hemolymph entering the thorax from the abdomen and warm hemolymph returning to the abdomen. There is also a thoracic heat exchanger consisting of a vertical hairpin loop of the aorta in the thoracic muscle. The sophisticated thermoregulatory system of *Eupsilia* is contrasted with the winter-flying moth *Operophtera*, which thermoconforms. This small (<10 mg) geometrid moth has a T_{th} only a few degrees above T_a even at 0° C. It is able to fly at such low temperatures because of its low energy cost of flight; its wing loading per mass is only 3.2 mg cm^{-2}, compared with 43 mg cm^{-2} for *Eupsilia*. *Operophtera* does not appear to have any enzymatic/metabolic specializations of the thoracic muscle to facilitate functioning at low T_a.

The bumblebee has a petiolar countercurrent heat exchange that is similar to that of the winter-flying moth *Eupsilia* (Figure 5–43B). This minimizes heat loss from the thorax to the abdomen at low T_a but can be bypassed at high T_a to dissipate thoracic heat and prevent an excessive increase in T_{th}.

Many moths warm up prior to flight; they elevate T_{th} by shivering contractions of thoracic flight muscle until it is high enough to sustain flight. Bumblebee flight muscle has an equivalent capacity to mammalian nonshivering thermogenesis. The flight muscle contains two enzymes, phosphofructokinase (PFK) and fructose bisphosphatase (FBPase). PFK catalyzes the phosphorylation of fructose-6-PO_4 to fructose-1,6-diPO_4, and FBPase catalyzes the reverse reaction (Hochachka and Somero 1984). The net effect of this futile cycle is hydrolysis of ATP to ADP + P_i. This futile cycle can be initiated at low muscle temperatures (<30° C) to release heat and

warm the thorax. When the muscles are sufficiently warm, the intracellular Ca^{2+} concentration is elevated; this inhibits FBPase and eliminates the futile thermogenic cycle.

The sphinx moth *Manduca* precisely regulates T_{th} at about 38° to 40° C while flying by using the abdomen as a heat sink at high T_a. Heat exchange between the thorax and abdomen is controlled by the nervous system. The abdominal heart responds to high T_{th} by increasing its pulsations, circulating hemolymph into and out of the thorax. Ligating the blood vessel compromises T_{th} regulation at high T_a.

A variety of other insects regulate a high body temperature by metabolic heat production during flight or while walking. For example, dung beetles can maintain a high T_{th} of 38° to 42° C while flying and rolling dung balls; tropical beetles can have T_{th} 4° to 16° C higher than T_a (the largest beetles have the highest $T_{th} - T_a$) during terrestrial activity; scarabid and cerambycid beetles can endogenously raise T_{th} by 5° to 7° C above T_a. The elephant beetle *Megasoma* is a large beetle (10 to 35 g), which can endothermically maintain T_{th} above T_a independent of locomotor activity, by a cyclic elevation of VO_2 rather than a sustained elevation of locomotory VO_2. Worker honeybees and incubating queen bumblebees also maintain homeothermy by nonlocomotory activity. Honeybees have the physiological capacity to elevate metabolic rate at low T_a and thereby maintain a high temperature of the hive or of a bee cluster. The relationship between VO_2 and T_a is essentially the same as that observed for an endothermic mammal or bird, and metabolic rate increases with cluster mass in the same fashion, and at an intermediate level, as the VO_2 of mammals and birds (Southwick and Heldmaier 1987).

Plants

Endothermy is not restricted to animals. Some plants produce sufficient metabolic heat to raise their floral temperature significantly above T_a. For example, the inflorescence of *Philodendron* can raise its temperature to 38° to 46° C at T_a of 4° to 39° C, by metabolic heat production of male sterile flowers (Nagy et al. 1972). The voodoo lily elevates its flower temperature by up to 22° C above T_a to volatilize its putrescent odor and attract insect pollinators. This is followed by a second phase of elevated (but lower) temperature when pollen is shed to warm the insect pollinators (Raskin et al. 1987). Some plants that flower in the snow also have a marked thermoregulatory capacity.

Evolution of Endothermy

We have seen essentially two different patterns of endothermy. Some large reptiles and fish, and many small insects, are endothermic as a consequence of activity, i.e., swimming, flying, or walking. Their metabolic rate is not regulated at varying T_a to maintain T_b constant. Homeothermy, if it is achieved by these animals, is by the physiological regulation of heat loss. In contrast, birds and mammals, brooding pythons, and some insects are endothermic by virtue of their physiological regulation of heat production (by shivering and nonshivering thermogenesis). The physiological regulation of heat loss is much less important.

The endothermic strategy of active insects and fish apparently evolved because locomotion produced sufficient metabolic heat that thermoregulatory strategies could be subsequently evolved. Elevated locomotory metabolism could well have preceded the evolution of thermoregulation. How did the latter endothermic strategy of birds and mammals evolve? It has also been suggested to have been the consequence of sustained activity, but there are several, although inconclusive, arguments against this. Birds and mammals rely on nonshivering thermogenesis, not shivering, for thermoregulation, except when cold stressed. Did nonshivering thermogenesis independently evolve as a precursor to endothermic homeothermy in birds and mammals, or did it independently evolve after shivering thermogenesis as an alternative mechanism? We have already seen that activity does not effectively substitute for thermoregulatory heat production in mammals, and so the hypothesis for the evolution of the endothermic strategy of birds and mammals from activity-derived metabolic heat is not compelling.

There are other hypotheses for the evolution of endothermy by mammals and birds. For example, mammalian homeothermy may have evolved in two steps (Crompton, Taylor, and Jagger 1978). First, small mammals (30 to 40 g) invaded the nocturnal niche. They regulated T_b at only about 25° to 30° C and their ($T_b - T_a$) gradient was probably only 10° C or less, so they did not require a marked capacity for thermogenesis. Tenrecs, which are nocturnal and have a low VO_2 and low T_b, may be an example of this thermal strategy. The second evolutionary step was a consequence of these nocturnal animals invading a diurnal niche; their T_b was raised to about 38° to 40° C to avoid the need to evaporatively dissipate water for thermoregulation of a low T_b (25° to 30° C) when subjected to a radiant heat load.

But, when and how did the mammalian metabolic machinery and insulation evolve? The metabolic rate of primitive mammals is the same as that of advanced mammals at equivalent T_b, i.e., the difference between "primitive" and "advanced" mammals is their T_b not their basic metabolic machinery (see Chapter 4). Why do living primitive mammals (including tenrecs) have essentially the same metabolic capacity and insulation as advanced mammals, if they represent the initial stage in the evolution of endothermy?

A second and very different scenario for the evolution of endothermy in mammals is the conversion of inertial homeothermy to endothermic homeothermy (McNab 1978). This theory begins with the reasonable assertions that the large reptilian ancestors of mammals were inertial homiotherms and the first endothermic mammals were small (shrew-sized). Homeothermy could only be maintained during this progressive reduction in size from reptile to mammal by the substitution of endothermic homeothermy for inertial homeothermy. This theory has the attraction that both insulation and increased basal metabolism could evolve gradually as body mass slowly declined from large reptiles to smaller mammals.

Advantages of a Constant Body Temperature. There are obvious advantages to endothermy and a constant T_b. An increased rate of enzymatic catalysis is a fundamental selective advantage to a higher body temperature. Enzymes can be adapted to function at low temperatures, but catalytic rates are nevertheless higher at elevated temperatures. A high T_b means that force and velocity of muscle contraction, and activity metabolic rate, are greater than at low T_b. A constant T_b allows enzymes to always be at their optimal temperature for catalysis. A high and stable T_b means that activity can be sustained irrespective of T_a, and so cold environments (nocturnal, high altitude, and latidude) can be exploited better by endotherms than ectotherms.

There are also costs to endothermic homeothermy. The principal disadvantage is the high energy expenditure for thermoregulation during periods of inactivity and low T_a. Endotherms must expend a greater fraction of their energy turnover on respiration rather than production (see Chapter 4).

There is an obvious evolutionary trend for the T_b of endothermic homeotherms to be regulated at successively higher values. This trend is apparent among mammals (e.g., monotremes and edentates have T_b of 30° to 32° C; cf. primates and lagomorphs with T_b of 38° to 39° C) and birds (e.g., ratites, 38° C; cf. passerines, 42° C). Ectothermic thermoregulators also tend to have similar, high T_b levels while thermoregulating, e.g., many lizards, 38° to 39° C; walking beetles and flying insects, 35° to 40° C. Why have these diverse thermoregulating animals evolved preferred T_b values in this general range of 35° to 40° C?

One disadvantage of a low preferred T_b is that it is more likely for T_a to approach or exceed T_b, thereby requiring evaporative cooling for T_b regulation. A higher T_b will minimize the likelihood of thermal stress. A higher T_b also increases the catalytic rate of reactions, up to a point. Enzymes can be adapted to higher temperatures (even up to 80° to 90° C) and so the ultimate limit to enzymatic/protein function is certainly not 40° to 45° C, even in higher eukaryote animals. What determines the maximum tolerable T_b? Why haven't animals evolved preferred T_b considerably higher than 35° to 40° C? Will they evolve even higher $T_{b,\text{pref}}$ values in the future millennia?

There are a number of disadvantages to a T_b that is too high. If $T_b \gg T_a$ a marked endogenous heat production is required to regulate T_b. If T_b did decline close to T_a (e.g., daily torpor) then this reduction in T_b would dramatically compromise the structure and function of enzymes and membranes. Maximal metabolism during torpor might be so reduced that arousal to a high T_b would be impossible, or at least energetically costly and slow. A very high T_b would require a correspondingly high energy acquisition. The physiological advantages of having a T_b of 50° C might not be sufficient compared to having a T_b of 40° C to justify the additional energy demands.

Other types of arguements have been offered to explain why T_b's often fall in the range 35° to 40° C. At about 37° C, any change in temperature will alter the free enthalpy of activation (ΔH^*) and free entropy of activation (ΔS^*) in an offsetting fashion, so that the free energy of activation (ΔG^*) is approximately compensated to a constant value (Hochachka and Somero 1984). The temperature at which ΔH^* and ΔG^* exactly offset each other is the compensation temperature (see below). Compensation temperatures for various enzymes are often about 35° to 55° C. Thus, the preferred T_b for endothermy may be adapted to minimize the overall effects of temperature on enthalphy and entropy for activation.

A less compelling arguement for the T_b range 35° to 40° C is provided by thermodynamic properties of water (Calloway 1976). A temperature of 37° C is consistent with some thermodynamic properties of water, e.g., the specific heat of water is minimal

at about 35° C; 38.5° C is the halfway temperature between the temperature of minimal thermal expansivity (4° C) and maximum (100° C); 40° C is the halfway temperature for kinetic reaction rates between 0° and 100° C. However, the physiological significance to an animal of such minimal or halfway temperatures is not clear. Even less compelling are observations such as the difference between the freezing and boiling points of water, divided by e (2.718) is 36.8° C, and the freezing point of water (in °K) divided by e^2 is 37.0° C.

Fever

Mammals, a wide variety of other vertebrates, and many invertebrates have a **fever** response (Table 5–16). Fever is an important and apparently general response of animals in which the thermoregulatory setpoint temperature is elevated by endogenous and exogenous pyrogens. For example, mammals generally have a rapid increase in T_b after the administration of bacterial toxins. Fever increases the hypothalamic setpoint and also the setpoint for onset of cutaneous vasodilation (for heat dissipation) and shivering (for heat production; e.g., rabbit). The hypothalamic thermostat is thought to be reset by a small protein, interkeukin, that is released from white blood cells in response to a variety of pathogens, such as bacteria and viruses.

Fever presumably has beneficial effects, especially as it is such a phylogenetically diverse phenomenon (Kluger 1979). The increase in T_b may enhance the activity of the immune system (see Chapter 15), e.g., the mobility and activity of white blood cells, stimulation and effect of interferon production, and activation of T-lymphocytes. Lizards (*Dipsosaurus*) injected with bacterial pyrogens have a higher survival at higher T_b (42° C) compared with lizards at lower T_b (e.g., 40°, 38°, 36°, and 34° C), suggesting that the higher T_b is advantageous.

Cryogens have the opposite effect as pyrogens, i.e., they lower the thermoregulatory setpoint. Mammals, including man, produce endogenous cryogens that induce a mild and transient hypothermia if injected into other mammals. For example, injection of human cryogens (present in the urine) can decrease the T_b of rabbits by 0.5° C.

TABLE 5–16

Effects of fever on body temperature (T_b) of invertebrates and vertebrates.

	Normal Preferred T_b	Fever T_b	ΔT
Dog *Canis*	38.2	39.4	1.2
Monkey	38.9	40.1	1.2
Rabbit	39.5	40.8	1.3
Beetle *Onymacris*	33	34.5	1.5
Desert iguana *Dipsosaurus* HBTS	41.0	42.7	1.7
Chimpanzee *Pan*	38.3	40.0	1.7
Pigeon *Columba*	39.7	41.5	1.8
Crayfish *Cambarus*	22.1	23.9	1.8
Bluegill sunfish *Lepomis*	30.1	32.2	2.1
Desert iguana *Dipsosaurus* LBTS	37.4	39.6	2.2
Large-mouth bass *Micropterus*	29.6	31.9	2.3
Frog *Hyla*	25.5	27.9	2.4
Tadpole *Rana*	28.5	31.2	2.7
Cockroach *Gromphadorhina*	32.3	35.9	3.6
Man *Homo*	37.4	41.3	3.9
Shrimp *Penaeus*	31	35.5	4.5
Lobster *Homarus*	16	20.7	4.7
Frog *Rana*	25.0	30.3	5.3
Horseshoe crab *Limulus*	27.0	33.0	6.0
Leech *Nephelopsis*	20.5	30.0	9.5
Scorpion *Androctonus*	24.8	28.8–39.8	≤15
Scorpion *Buthus*	25.1	25.1–43.1	≤18

Acclimation and Acclimatization

Temperature affects the rates of most physical, biochemical, and physiological functions, generally with a Q_{10} of 2 to 3. However, the biochemistry and physiology of animals are not necessarily at the mercy of the thermal environment and their T_b because biochemical and physiological rates can be adjusted to compensate for variations in temperature. Such compensation for temperature is called **acclimatization** if it occurs in nature, and **acclimation** if it is induced in the laboratory. For example, the $T_{b,pref}$ of many fish is lower during the colder parts of the year than during the warmer parts of the year (acclimatization). Fish kept in the laboratory at various water temperatures show a similar decrease in $T_{b,pref}$ in the colder water (acclimation). Many biochemical and physiological processes show thermal acclimation/acclimatization, e.g., enzyme reaction rates, heart rate, metabolic rate, respiratory rate, preferred T_b, CT_{min}, and CT_{max}. There is also

a suite of morphological, enzymatic, and physiological adaptations for thermal acclimation in plants (Chabot 1979).

In general, acclimation and acclimatization maintain similar rates at varying temperatures, i.e., the rate is the same for a cold-acclimated animal at its T_a and a warm-acclimated animal at its higher T_a. However, there are a number of different patterns in acclimation/acclimatization (Precht 1958; Prosser 1958). The types of acclimation/acclimatization recognized by Precht are summarized in Figure 5–44A. Type 2, or perfect acclimation, results in a rate after thermal acclimation that is exactly the same as the initial rate. We might expect this to be the most prevalent, and ideal, type of acclimation but there are many circumstances in which perfect acclimation is not optimal. Type 3, or partial, acclimation occurs if an acclimation response partially returns the rate to the preacclimation value. There are many examples of incomplete acclimation, perhaps reflecting the biochemical difficulty in completely compensating for a change in temperature, or the physiological unnecessity for perfect acclimation. Type 4 is no acclimation; there is a lack of any acclimation response to a change in temperature. Type 5 acclimation is inverse acclimation. Such an acclimation pattern might be adaptive, for example, during winter dormancy when the metabolic rate is depressed by the lowered temperature and is further depressed by the inverse acclimation. For type 1, or over-acclimation, the rate after acclimation is higher than the initial rate after acclimation to a

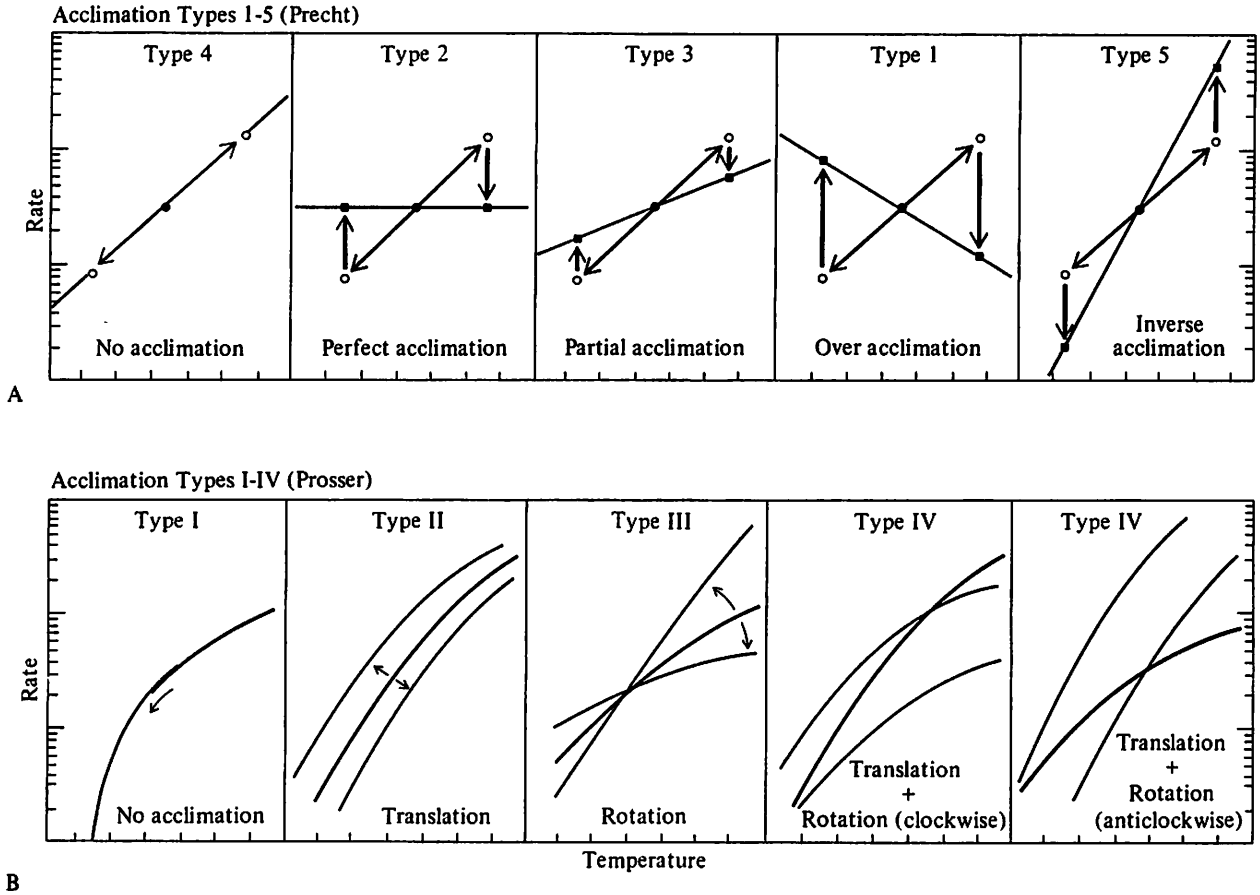


FIGURE 5–44 (A) Patterns of acclimation as defined by Precht. The solid circles indicate the initial rate/temperature and the open circles indicate the initial, acute change in rate/temperature (thin arrows); the solid squares and colored lines indicate the rate/temperature after thermal acclimation (thick arrows). **(B)** Patterns of acclimation as defined by Prosser. The solid lines indicate the initial rate-temperature curve; the colored lines indicate the rate-temperature curve after thermal acclimation. *(Modified from Precht 1958; Prosser 1958.)*

lower temperature (or lower than the initial rate after acclimation to a higher temperature). Such an acclimation response might be useful, for example, in limiting the effect of high temperature ($>T_{b,pref}$) on reaction rates.

The acclimation scheme of Prosser (Figure 5–44B) considers the effect of acclimation over a range of temperatures, whereas the Precht classification scheme considered the change between only two temperatures. The relationship between rate over a variety of temperatures may be unaltered by acclimation (Type I, no effect ≡ Precht Type 4), may move up or down (Type II, translation ≡ Precht Type 1, 3, 4, or 5), may rotate about a constant rate at one temperature (Type III), or may translate and rotate (Type IV).

Many invertebrates show thermal acclimation (Cloudsley-Thompson 1970). The CT_{max} of the earthworm *Pheretima* increases by 0.3° C per 1° C rise in acclimation temperature. The slug *Arion circumscripta* shows metabolic acclimation. Many arthropods also show various types of thermal acclimation. The CT_{min} and CT_{max} of two isopods are influenced by acclimation temperature (Table 5–17). Thermal acclimation may occur quite rapidly (<24 hr). Cockroaches (*Blatella*) transferred from a warm to a cold environment show almost complete thermal acclimation within a few to about 24 hours for transfer from 25° to 15° C, but require longer to acclimate from 35° to 25° and 35° to 15° C (Figure 5–45A). Thermal acclimatization may also occur on a latitudinal or altitudinal gradient. For example, there are seasonal changes in the type of the third chromosome in the Californian fruit fly *Drosophila pseudoobscura* (the SI, AR, and CH types). At 30° C, ST-type pupae have a high survival and ST-adults have a greater longevity than CH flies. A laboratory population of flies showed a shift in frequency to 70% ST when transferred from 17° to 25° C. Northern populations (i.e., cooler climates) of the European fruit fly *D. funebris* are more resistant to lower temperatures, and southern populations (i.e., warmer climate) are more resistant to higher temperatures. Eggs of the tortricid moth *Acrolite* from eastern Norway accumulate more glycerol (for freezing tolerance) than do eggs of western Norway moths that experience a milder climate. Orbatid mites from West Africa have a higher CT_{max} (37° C) than mites from North America (30° C).

TABLE 5–17

Critical thermal minimum (CT_{min}) and critical thermal maximum (CT_{max}) temperatures for the terrestrial isopods (*Porcellio laevis, Armadillidum vulgare*), as a function of acclimation temperature. *(Data from Edney 1964.)*

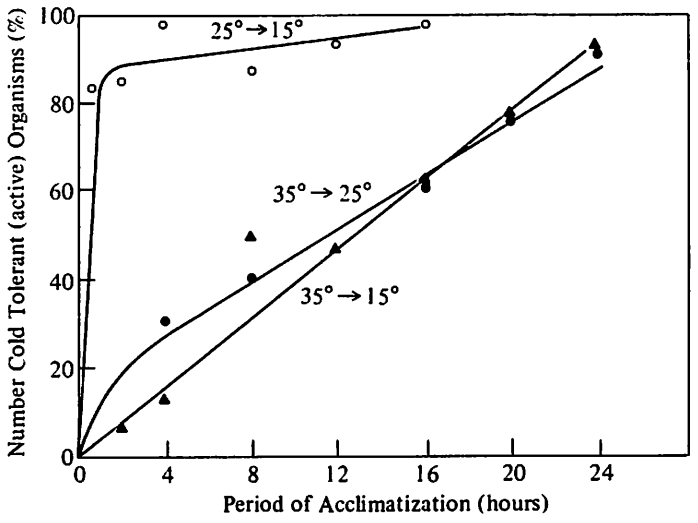
Acclimation Temperature	*Porcellio laevis*		*Armadillidum vulgare*	
	CT_{min}	CT_{max}	CT_{min}	CT_{max}
10° C	−2.4° C	37.4° C	−2.7° C	38.3° C
30° C	5.5° C	41.6° C	3.0° C	41.6° C

Fish generally show a substantial metabolic acclimatization, e.g., comparing species from cold and warm climates, and also for individuals acclimatized (or acclimated) to various temperatures. For example, temperate fish have a similar metabolic rate as arctic and antarctic fish despite marked differences in their ambient temperature (Figure 5–45B). However, tropical fish tend to have higher metabolic rates than temperate fish, showing more of a Q_{10} effect than an acclimatory compensation. There is also marked acclimation in both CT_{min} and CT_{max} of fish. For example, the CT_{max} of the salmon *Oncorhynchus keta* varies from about 22° to 24° C at acclimation temperatures of 0° to 40° C; CT_{min} varies from 0° to 7° C. A temperature polygon showing similar changes in CT_{min} and CT_{max} for *O. nerka* was described in Chapter 2.

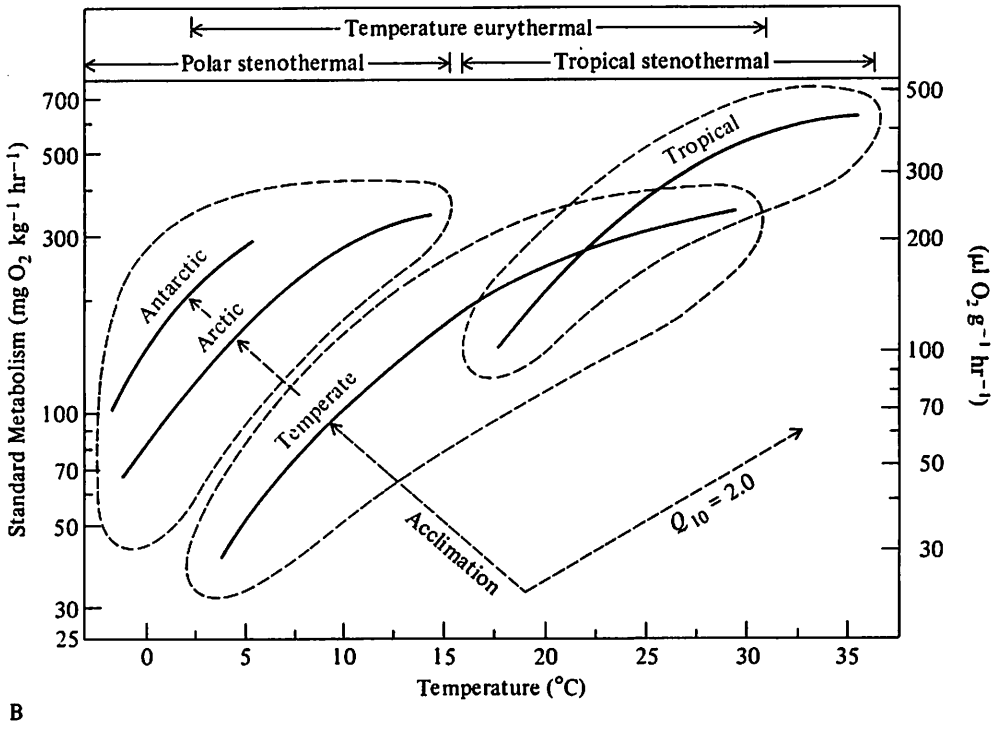
Amphibians and reptiles generally show thermal acclimation of metabolic rate, CT_{min} and CT_{max}. For example, there is thermal acclimation of CT_{max} in temperate and tropical anuran amphibians, although CT_{max} is higher in the tropical species at equivalent acclimation temperatures. There is also a general trend for CT_{min} to decrease for amphibians at higher (colder) latitudes.

Rates of acclimation are quite variable but generally follow a hyperbolic curve with complete acclimation in two to four days. The thermal response ratio (TRR) is the change in CT_{max} per change in acclimation temperature ($\Delta CT_{max}/\Delta T_a$). The TRR and the time for 50% acclimation (1/2 AT) are commonly used to describe the time course and magnitude of acclimation. For amphibians, the TRR varies from about 0.07 to 0.44 and 1/2 AT varies from 0.12 to 2.8 days (Table 5–18). Both CT_{max} and CT_{min} of reptiles shows thermal acclimation. In turtles, there is a daily variation in CT_{max}. The $T_{b,pref}$ of lizards can also vary with time of day or season, with age, and with hormonal and physiological state.

Endotherms also show thermal acclimation of many physiological and biochemical variables, although in response to variation in T_a rather than T_b. For example, acclimation responses include



A



B

FIGURE 5–45 (A) Time course for acclimation of cold tolerance in the cockroach *Blatella* from high ambient temperature (25° or 35° C) to a lower ambient temperature (25° or 15° C). **(B)** Thermal acclimation of metabolic rate for polar, temperate, and tropical fish. *(From Colhoun 1960; modified from Brett and Groves 1979.)*

nonshivering thermogenesis, fur and feather thickness and color, nerve conduction velocity, ability to become torpid and $T_{b,crit}$ during torpor, the melting point of lipids in hypothermic limb extremities, sweating rate, and lowering of basal metabolic rate.

Biochemical Adaptations to Temperature

Thermal acclimation is an important physiological phenomenom that allows cold-adapted animals to have "normal" biochemical and physiological rates

TABLE 5–18

The magnitude and rate of thermal acclimation in critical thermal maximum (CT_{max}) for salamanders. The magnitude of thermal acclimation in CT_{max} is indicated as the thermal response ratio (change in CT_{max} per change in acclimation temperature $\Delta CT_{max}/\Delta T_{acc}$); the rate of acclimation is indicated as the half time for attainment of the new CT_{max} ($\frac{1}{2}$ AT; days). *(Data from Claussen 1977.)*

	Acclimation Temperature °C	Thermal Response Ratio $\Delta CT_{max}/\Delta T_{acc}$	Half Time $\frac{1}{2}$ AT
C. alleghaniensis	5→25/25→5	0.20/0.23	2.80/2.15
N. maculosus	5→25/25→5	0.21/0.16	1.54/1.18
N. viridescens	5→25/25→5	0.16/0.17	0.17/2.24
C. multidentatus	5→25/25→5	0.17/0.19	0.39/1.00

at low temperature, compared to warm-adapted animals. There are a number of adaptive changes in both enzyme structure and function, and lipid membrane structure and physical properties, that occur during thermal acclimation.

Enzymes. There are a number of potential strategies to manipulate enzyme catalytic rate and achieve thermal acclimation. These include altering the following: (1) enzyme concentration; (2) substrate concentration; (3) catalytic efficiency of enzymes; and (4) the intracellular environment, e.g., ionic concentration and pH. Enzyme concentration and catalytic efficiency are generally the more important adjustments during thermal acclimation (Hochachka and Somero 1984).

Many ectotherms and endotherms show acclimatory changes in enzyme concentration. The metabolic rate of cold-acclimated ectotherms can be equivalent to that of warm-acclimated ectotherms if there is an increased concentration of the key, rate-limiting enzymes. Not all enzyme concentrations have to be increased, just those for enzymes of reactions that limit the overall reaction rate. The key enzymes for aerobic metabolism may show a marked temperature compensation (Hazel and Prosser 1974). The mitochondrial protein content of eel liver increases with cold acclimation, from <4 mg g^{-1} at 25° C to 6 mg g^{-1} at 7° C; eels also show partial thermal acclimation in VO_2 to lowered temperature (Wodkte 1973). However, the increase in mitochondrial protein does not result in a proportional maintenance of VO_2 because the specific activity of the protein is reduced at the lower temperature, and so the total VO_2 of the liver mitochondria is lower at 7° C than 25° C. The cytochrome oxidase activity of goldfish skeletal muscle of 45 μmol sec^{-1} mg $protein^{-1}$ at an acclimation temperature of 5° C is higher than the value of 20 at an acclimation temperature at 25° C. An adaptive change in cytochrome oxidase concentration might be inferred from these results, but it is important to appreciate that a change in catalytic rate is not direct evidence for a change in enzyme concentration *per se*. Nevertheless, there are demonstrated changes in cytochrome oxidase concentration for the green sunfish during thermal acclimation (Sidell 1977), and so we can conclude that changes in enzyme concentration are sometimes one of the mechanisms for thermal acclimation. However, the general utility of achieving thermal acclimation by adjustment of enzyme concentrations is questionable. Synthesizing high concentrations of enzyme to compensate for its thermally induced catalytic inefficiency is not necessarily an optimal solution. For example, glycolytic enzymes may show little, or even inverse, acclimation in concentration.

Substrate concentration can significantly influence reaction rates and their temperature dependence. Low substrate concentrations reduce the Q_{10} effect, often substantially below the Q_{10} for V_{max}. For example, the Q_{10} of LDH varies from <1.5 (at <0.1 mM pyruvate) to >1.5 (at >0.8 mM pyruvate) for fish and lizards. The Q_{10} for pyruvate kinase of a crab varies from <2.0 (at <2.0 mM PEP) to >3 (at >0.5 mM PEP). However, substrate concentration tends to be remarkably similar among different species and adjustment in K_m is a more important mechanism for maintaining catalytic rates.

The catalytic efficiency of enzymes is generally temperature dependent. However, homologous enzymes from different individuals, or species, can counteract the effect of temperature on catalytic rate by variation in their free energy of activation (ΔG^*). For example, cold-adapted enzymes may have a lower ΔG^* than warm-adapted enzymes to minimize the change in catalytic efficiency. Reaction velocity is not so dependent on temperature if $\Delta G^*/T$ is fairly constant, since

$$V = \frac{kT}{h} e^{-\Delta G^*/RT} \qquad (5.21)$$

where k is the Boltzmann constant and h is Planck's constant. For example, the $\Delta G^*/T$ increases slightly for cold-adapted enzymes (0.216 for LDH of the ice fish *Pagothenia* compared with 0.194 for the rabbit) so that V decreases for the cold-adapted enzyme,

but not by as much as it would if ΔG^* did not decrease. For Mg^{2+}-Ca^{2+} myofibrillar ATPase, the ΔG^* declines more dramatically with lowered temperature and the $\Delta G^*/T$ decreases for the low temperature enzyme; the V is slightly higher for the cold-adapted species whereas it would have been much lower if the ΔG^* had not been lower for the cold-adapted enzyme. Thus, modification in ΔG^* can have profound effects on thermal sensitivity of enzymes.

How is ΔG^* varied for different enzymes? The structures of substrate molecules and cofactors are invariant, and the chemistry of the reaction at the active site is also likely to be invariable for homologous enzymes. It is enzyme structure that is modified (Hochachka and Somero 1984). The flexibility of an enzyme's structure reflects the degree of covalent and noncovalent bonding between the constituent amino acids. Enzyme catalytic efficiency is related to its structural stability, due to weak bonding. Weak bonds have a low free energy of formation (Van der Waals forces, -4.2 kJ mole^{-1}; hydrogen bonds, -20.9; ionic bonds -20.9; hydrophobic interactions, $+8.4$) and are susceptible to thermal perturbation. Covalent bonds, in contrast, have a high free energy change (e.g., *C*—*C*, -350 kJ mole^{-1}; *S*—*S*, -210) and confer a considerable structural stability at high temperatures. Increased thermal stability by more weak bonding decreases the flexibility of enzymes, hence decreases their catalytic and regulatory capacity. This is indicated by the strong correlation between activation enthalpy (ΔH^*) and activation entropy (ΔS^*) for homologous enzymes (Figure 5–46). This graph is called a compensation plot because it shows that an increase in ΔH^* is compensated by an increase in ΔS^* ($\Delta G^* = \Delta H^* - T\Delta S^*$). The compensation temperature is the slope of the compensation plot; it indicates the temperature at which the enthalpy change for homologous enzymes is exactly balanced by entropy change. Compensation temperatures are generally 25° to 60° C.

Temperature not only affects the reaction velocity but also affects the Michaelis-Menten coefficient

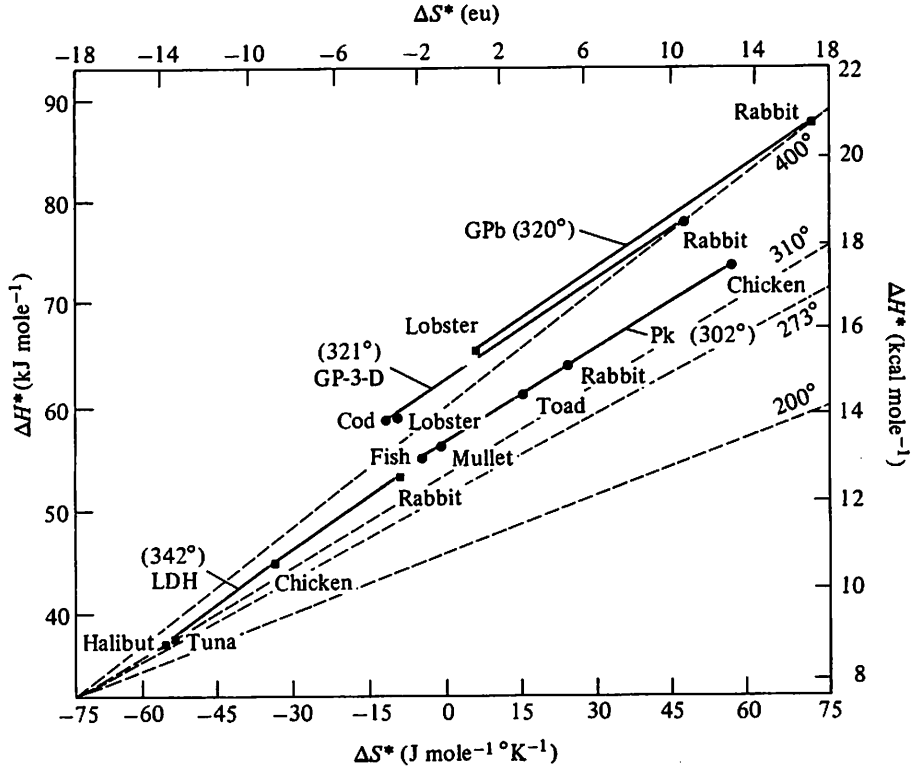
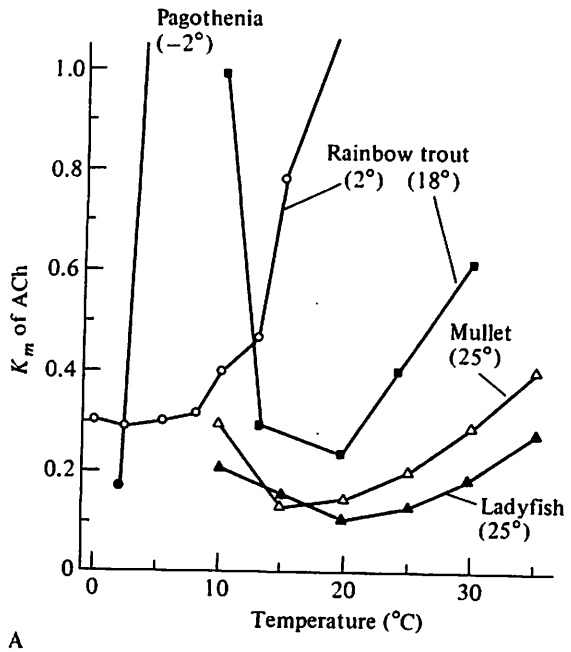


FIGURE 5–46 Compensation plots (ΔH^* as a function of ΔS^*) for homologous forms of the enzymes pyruvate kinase (Pk), glycogen phosphorylase b (GPb), lactate dehydrogenase (LDH), and glyceraldehyde-3-phosphate dehydrogenase (GP-3-D) from various vertebrates. The slopes of the lines (compensation temperature, °K) are indicated for each enzyme. The dotted lines indicate theoretical relationships for the indicated compensation temperatures. *(Modified from Somero and Low 1976.)*

(K_m), i.e., the affinity of the enzyme for its substrate. There may be an optimal temperature at which K_m is minimal and catalytic efficiency is maximal, e.g., acetylcholinesterase from trout brain (Figure 5–47A) or K_m continues to decline at lower temperatures, e.g., LDH of bluefin tuna (Figure 5–47B). In the former example, the increase in K_m at temperatures below the optimum value (about 20° C) will result in high Q_{10} values since a decrease in temperature will not only decrease reaction rate by the normal Q_{10} effect but also decreases the affinity of the enzyme for its substrate; this is called negative thermal modulation. The effect of elevated temperature on reaction rate is minimized by an increase in K_m; with elevated temperature (apparent for both trout brain AChE at $T > 20°$ C, and bluefin tuna LDH). This is positive thermal modulation and keeps the Q_{10} low.



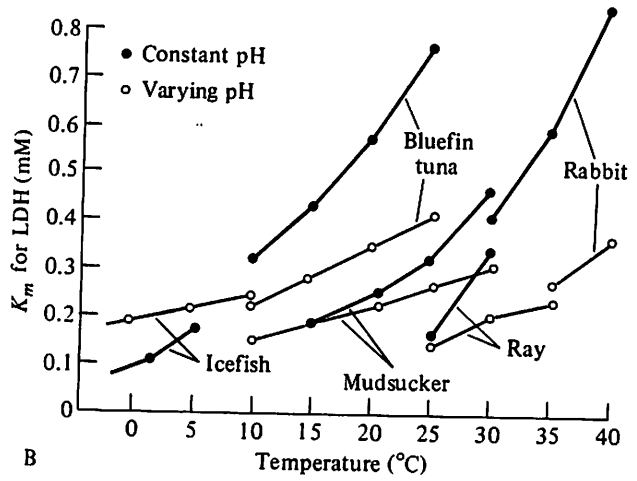


FIGURE 5–47 (A) Effect of temperature on the K_m of acetylcholine for acetylcholinesterase (AChE) of fishes from varying thermal environments; the trout has two isozymes adapted to 2° C and 20° C. **(B)** Effect of temperature on K_m for pyruvate of LDH from several vertebrates adapted to varying temperatures, at constant pH (pH = 7.4; solid circles) and at a pH buffered by imidazole to conform to the thermal effect on the neutral point for water (open circles). *(From Hochachka and Somero 1984; Yancey and Somero 1978.)*

Cold-adapted enzymes tend to have a similar K_m as warm-adapted enzymes at their respective temperatures; this requires a significant shift in the K_m temperature curve. For example, the congeneric barracuda (*Sphyraena argentea, S. lucasana, S. ensis*) occur in temperate, subtropical, and tropical areas of the western coast of North America, respectively. The kinetic properties (K_m, k_{cat}) of the muscle LDH of these fish vary when measured at the same temperature (e.g., 25° C) but are similar at the temperature appropriate to their natural environmental temperature (Table 5–19). The k_{cat} is the turnover number per active site, or moles of substrate converted per mole enzyme per unit time.

Adaptive changes in the thermal sensitivity of homologous enzymes from different populations, or different species, may reflect allelic variation in the structure of the enzyme; such allelic variants are called **allozymes**. A convincing example of allozymic thermoadaptation is the heart-type LDH of the fish *Fundulus* (Place and Powers 1979). The LDH_a gene from southern populations is replaced progressively by the LDH_b gene in more northern populations. The ratio of k_{cat}/K_m is an *in vivo* measure of catalytic efficiency. This ratio is maximal at 20° C for the cold-adapted LDH_b and at 30° C for the warm-

TABLE 5–19

Kinetic parameters at 25° C for LDH of three barracuda *(Sphyraena* spp) from different thermal environments, and the kinetic parameters at the normal temperature midrange (T_M) for each species. *(Data from Graves and Somero 1982.)*

	S. argentea	*S. lucasana*	*S. ensis*
T_M	18° C	23° C	26° C
K_m at 25° C	0.34 mM	0.26 mM	0.20 mM
K_m at T_M	0.24 mM	0.24 mM	0.23 mM
k_{cat} at 25° C	893 sec^{-1}	730 sec^{-1}	658 sec^{-1}
k_{cat} at T_M	667 sec^{-1}	682 sec^{-1}	700 sec^{-1}

adapted LDH_a. The skeletal muscle LDH of congeneric barracuda provide a similar example of allozymic variation in enzyme catalytic properties.

Some examples of thermal adaptation for enzymes of individual animals can reflect variation in the thermal properties of different forms of the enzymes. For example, rainbow trout (*Salmo gairdneri*) acclimated to varying temperatures have different forms of acetylcholinesterase (e.g., minimum K_m at 2° and 18° C; Figure 5–47A). The 2° C AChE has a low K_m at low T_a whereas the 18° C AChE has a low K_m at high T_a. These enzyme variants of individual animals are called **isozymes** because they represent variation in the multiple copies of the genetic code for the enzyme (e.g., LDH). The multiple isozyme strategy is not very common; most species do not have "cold" and "warm" isozymes, perhaps because of the additional genetic load of multiple copies of enzymes (the rainbow trout is tetraploid, rather than diploid, and therefore has essentially twice as much genetic material coding for enzymes). However, many species have various isozymes in different tissues or organs, e.g., muscle and heart LDH.

A final potential mechanism for compensation of thermal modulation of enzyme catalytic efficiency is modification of the intracellular environment in which the enzymes function. For example, there are changes in ionic concentration of intra- and extracellular fluids that accompany thermal acclimation (Behrisch 1973). The K_m of LDH from the yellowfin sole (*Limanda*) has a minimum value at 4° C (measured with no K^+ present). The yellowfin sole lives in water ranging in temperature from −1.86° C in winter to 4° to 5° C in summer, and so it might seem that its LDH functions at submaximal catalytic efficiency for most of the year. However, the K_m measured in 150 mM K^+ (a more physiologically relevant condition) has a minimum at about −2° to 0° C; the normal ionic K^+ concentration is required for optimal enzyme function.

Temperature has a marked effect on the pH of neutral water, and the pH of intracellular and extracellular body fluids (see Chapter 12). This pH temperature dependence provides significant stabilization of enzyme kinetics (e.g., K_m). For example, the K_m of muscle LDH has a marked temperature dependence when pH is kept constant at 7.4 by a phosphate buffer (Figure 5–46B). For many animals, such a constant pH is not physiologically relevant since pH increases at lower temperatures. There is a lesser dependence of K_m on temperature in an imidazole buffer, which mimics the *in vivo* temperature pH relationship for many animals.

The thermal stability of proteins is often implicated as a cause of thermal death. There is a correlation between the denaturation temperature for proteins (melting temperature, T_m) and the CT_{max}, but enzyme catalytic efficiency is likely to decline markedly and cause death well before proteins actually denature. Enzyme structure is generally quite flexible to maintain catalytic efficiency at low temperature, and so proteins may become too flexible at high temperature. The proteins of thermophilic bacteria are remarkably heat tolerant; some can tolerate 90° C *in vitro*. Their high stability may be due to increased ionic bond stabilization, reduced surface hydrophobicity and enhanced internal hydrophobicity, or increased covalent stabilization. The bacterium *Thermus* also contains thermoprotective polyamines that protect its proteins from thermal denaturation. There would appear to be only a limited capacity for animal proteins to decrease their thermal sensitivity because their catalytic functions would be reduced at lower temperatures.

Lipid Membranes. Biological membranes are essentially a bilayer of amphiphilic lipids (one end is polar and the other is nonpolar) such as phospholipids and sphingolipids (see Chapters 3 and 6). Phospholipids, for example, consist of two fatty acids (the nonpolar end) bound to a glycerol, and a phosphate with a polar headgroup such as choline or ethanolamine (the polar end). Associated with the lipid bilayer are a variety of structural and enzymatic proteins that may be an integral part of the membrane or located more peripherally on the surface.

The physical properties of lipids, and especially lipid bilayer membranes, are markedly influenced by temperature. Biological membranes exist in a "liquid-crystalline" state that is functionally intermediate between a rigid, solid lipid (e.g., lipids at low temperature) and a highly fluid lipid state (e.g., lipids at high temperature). The phospholipid extracts of a bacterial membrane (*E. coli*) clearly illustrate a phase transition between a low viscosity at high temperature to a high viscosity at low temperature Figure 5–48). The maintenance of a normal **homeoviscous** lipid state in biological membranes is essential to the functioning of the membrane.

One of the major variables contributing to the homeoviscous state is the fatty acid composition of the lipid bilayer. Fatty acids vary in both chain length and degree of double bonding of the carbon backbone. Shorter chain fatty acids are more liquid than longer chain fatty acids. Unsaturated fatty acids (with double bonds) are more liquid than

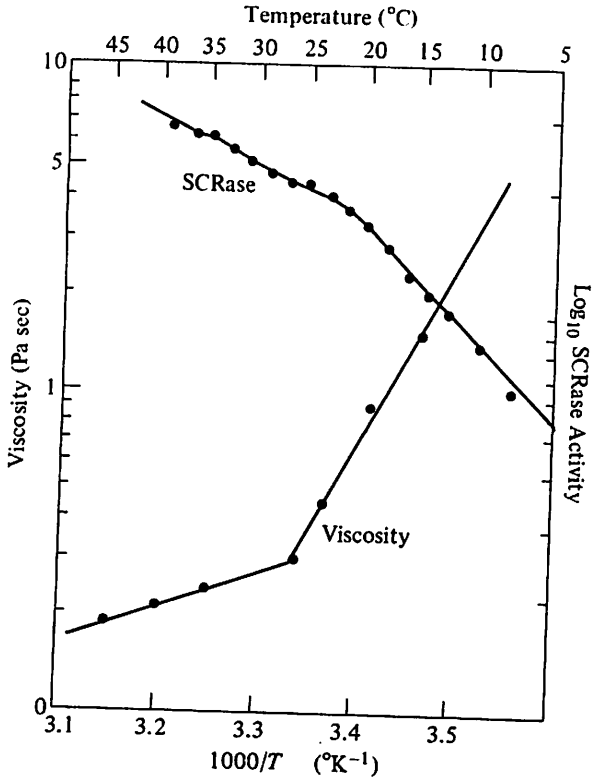


FIGURE 5–48 Effect of temperature (Arrhenius plot) on the viscosity of phospholipids extracted from a bacterium showing a phase transition at about 27° C, and on the activity of mitochondrial cytochrome C reductase (SCRase) from the guinea pig. *(Modified from Sinensky 1974; Geiser and McMurchie 1984.)*

TABLE 5–20

Effects of ambient temperature on the ratio of saturated to unsaturated fatty acids for three phospholipids (choline, ethanolamine, serine/inositol) isolated from brain lipid membranes of a range of vertebrates. *(Data from Cossins and Prosser 1978.)*

		Phospholipids		
	Temperature	*Choline*	*Ethanolamine*	*Serine/ Inositol*
Arctic sculpin	0°C	0.593	0.947	0.811
Goldfish	5°C	0.659	0.340	0.459
	25°C	0.817	0.506	0.633
Desert pupfish	34°C	0.990	0.568	0.616
Rat	37°C	1.218	0.651	0.664

saturated fatty acids because their "kinked" shape reduces stabilization of adjacent molecules. The lipid fluidity is correspondingly adapted to the normal environmental temperature.

The membrane lipids of animals and plants from varying ambient temperatures show changes in fatty acid composition that are adaptive for maintenance of the homeoviscous state (Table 5–20). The variation in fatty acid composition of membrane lipids is accomplished by regulating the chain length and polar head group composition or degree of saturation (see desaturase enzymes below). Rainbow trout acclimated to 20° C then cooled to 5° C have a decline in phosphatidyl choline level and an increase in phosphatidyl ethanolamine; there is a significant change in the PC/PE ratio within three days. The fatty acid composition of the nerve cord of an insect has a decreased level of three saturated fatty acids (myristic, pentadecanoic, and palmitic) at low temperatures, and increased content of three unsaturated fatty acids at high temperature (linoleic, eicosadienoic, and arachidonic); the fat body lipid does not show a corresponding temperature-dependent change in fatty acid content. There are extremely rapid changes (<12 hours) in head group composition for ectotherms that experience marked daily temperature fluctuations (Carey and Hazel, 1989).

Desaturase enzymes regulate the degree of unsaturation of fatty acids. The activity of desaturase enzymes can be rapidly modified in response to temperature changes, either by a direct thermal effect on their activity (high temperature inactivates the desaturase enzyme) or by a change in the membrane location of the desaturase (the active site is exposed in low fluidity membranes but hidden within the lipid bilayer in high fluidity membranes). For example, carp initially acclimated to 30° C have an enhanced desaturase activity and concentration after transfer to 10° C, and this results in the restructuring of the rough endoplasmic reticulum lipids within two days of cooling to 10° C (Wodtke, Teichert, and Konig 1986).

The homeoviscous state of a membrane has great significance to membrane-bound enzyme catalysis. Membrane fluidity would influence the catalytic activity of enzymes that must undergo conformational changes during catalysis, e.g., membrane-bound transport proteins. The fluidity of the lipid bilayer surrounding the enzyme clearly would influence the ability to undergo conformational change. Membrane fluidity may also influence the location and exposure of the active site. This concept agrees well with the observation that the activation energy (E_a) increases for many membrane-bound enzymes below a critical temperature; this is readily apparent

from Arrhenius plots (Figure 5–48). At temperatures below the breakpoint, the enthalpy of activation (ΔH^*) is increased dramatically; this is compensated to some extent by the increased activation entropy (ΔS^*) and so the free energy of activation ($\Delta G^* = E_a$) is not so much affected as is the ΔH^*.

Summary

Temperature is a measure of the average thermal motion of molecules. It dramatically affects reaction rate, as indicated by the activation energy (E_a) and Q_{10} values of biochemical and physiological rates. Heat exchange can occur by conduction, convection, radiation, and a change in state of water (evaporation/condensation or freezing/thawing).

Animals either conform to their thermal environment or thermoregulate. The T_b of ectothermic animals is determined by heat exchange from their environment; their metabolic heat production is negligible. Many ectotherms are thermoconformers. Aquatic ectotherms may thermoregulate by selecting water of the appropriate temperature. Terrestrial ectotherms can be accomplished thermoregulators by either basking in sunlight or gaining heat from the substrate by conduction. Endothermic animals thermoregulate by virtue of their endogenous metabolic heat production.

Conductive heat exchange depends on the thermal conductivity, area of contact, distance for heat transfer, and the temperature difference between the objects. The thermal conductivity of animal insulation (fur, feathers, and chitin hairs) is similar to that for still air; their resistance to heat exchange, and insulative value, depend on the thickness of the insulating layer. Subcutaneous fat has a low insulative value in air but is a better insulator in water than fur or feathers.

Convective heat exchange is essentially conduction across the boundary layer, in proportion to the temperature difference and convective heat transfer coefficient. Forced convection transfers heat more rapidly than free convection.

Net radiative heat exchange depends on the surface temperature of the animal, the average temperature of the surroundings, area, and emissivity. The radiative heat transfer coefficient is markedly dependent on temperature.

Evaporation dissipates about 2500 J g^{-1}; condensation releases the same amount of heat. Freezing releases about 334 J g^{-1}; whereas melting absorbs the same amount of heat.

The thermal environment of animals is complex. The operative temperature is the effective environmental temperature determined by conductive, convective, and radiative heat exchange. The standard operative temperature is the operative temperature with standardized convective conditions (usually free convection). These are better measures of the environmental temperature than is air temperature.

Ectotherms avoid freezing by behavioral avoidance or physiological adaptation. Strategies for physiological adaptation include freezing point depression by the accumulation of specific osmolytes (e.g., sugars, polyols), supercooling to temperatures below the freezing point without ice formation (often facilitated by specific accumulated osmolytes), inhibition of freezing by antifreeze proteins (a noncolligative effect), and tolerance of extracellular freezing. Ectotherms adapt to high temperatures by enhanced evaporative cooling or biochemical acclimation.

Endothermic mammals and birds regulate body temperature by control of their endogenous heat production. Body temperature is generally regulated at 35° to 42° C, depending on the taxonomy of the mammal or bird. Metabolic heat production is highest at low T_a, is minimal (basal) in the thermoneutral zone, and is elevated at high T_a. Endotherms have three strategies for survival in the cold: (1) they decrease heat loss, (2) they increase heat production, or (3) they decrease body temperature. The hypothermia may involve only the peripheral appendages (the core T_b is maintained; regional heterothermy), or there may be a reduced core T_b (temporal heterothermy). The core T_b may be decreased slightly, as in moderate hypothermia, or markedly depressed, as in torpor. Torpor is the abandonment of normal T_b thermoregulation, and T_b declines to near T_a. At low T_a the T_b is regulated at a minimal $T_{b,crit}$ value. Endotherms respond to high T_a by enhancing evaporative heat loss. The T_b may be increased (hyperthermia) to facilitate heat dissipation. Brain temperature is often regulated below core T_b to avoid nervous system disfunction; there is countercurrent heat exchange between warm arterial blood and cool venous blood returning from evaporative surfaces of the head (nasal cavity, skin, eyes).

The only living endothermic reptiles are some brooding female pythons, which shiver to regulate body and egg temperature relatively independent of T_a. Large reptiles (>100 kg) are homiothermic by virtue of their high mass and thermal inertia, and low thermal conductance. Their T_b can considerably exceed T_a because of passive constraints to thermal dissipation. Large dinosaurs may have been endothermic and regulated their T_b by physiological means, including control of metabolic heat production.

Large, active fish, such as tuna, sharks, and swordfish, are regional endotherms. Metabolic heat production of, for example, skeletal muscle is retained within tissues by vascular countercurrent heat exchange. Muscle, brain, eye, or visceral temperature can thus be maintained considerably above the ambient water temperature. The rate of metabolic heat production is not varied to regulate T_b. Rather, metabolic heat production is constant and the rate of heat loss is controlled.

Many flying, running, or walking insects are endothermic and regulate thoracic temperature. Generally, the rate of metabolic heat production is determined by the intensity and type of locomotion and is not controlled to regulate thoracic temperature. Thoracic insulation and countercurrent heat exchange between thorax and abdomen facilitate preflight warm up and thoracic temperature regulation during flight at low T_a.

Endothermy has evolved in a variety of animals. One possible scheme for the evolution of endothermy is the initial elevation of T_b by metabolic heat production from locomotion followed by the acquisition of insulation or vascular heat exchangers for further elevation and regulation of T_b. Alternatively, mammalian endothermy may have evolved during a change in niche from nocturnality to diurnality, or the progressive reduction in body size from large, inertially-homiothermic reptiles to smaller mammals.

The advantages of endothermy include optimal biochemical adaptation to the constant and high T_b, high muscle force, velocity and power expenditure, and independence of activity from ambient thermal conditions. The principal disadvantage of endothermy is the high metabolic cost for thermoregulation.

The T_b of endotherms is generally 35° to 42° C. This range presumably reflects the catalytic advantages of higher temperature for biochemical and physiological processes and the disadvantages of too high a T_b of excessive metabolic expenditure for thermoregulation and excessively high demands for energy consumption.

Endogenous and exogenous pyrogens increase the T_b of a variety of ectotherms and endotherms. For example, heliothermic lizards will select higher temperatures in a thermal gradient, and mammals will shiver to elevate T_b above the normal preferred temperature. The presumed selective advantage for the hyperthermia is an enhanced immune response and/or diminished viability of the infecting pathogen.

Biochemical and physiological reactions are thermally dependent but can acclimate (in the laboratory) or acclimatize (in nature) with prolonged exposure to differing temperatures. There are a number of patterns of thermal acclimation. Generally, the acclimation response is adaptive; the rate after acclimation to a new temperature is more similar to the initial rate than was the rate immediately after the temperature change. Many physiological processes show thermal acclimation, e.g., metabolic rate, respiratory rate, heart rate, preferred T_b, critical thermal maximum and minimum temperatures. There are a variety of biochemical mechanisms for thermal acclimation: changes in enzyme concentration, alteration of substrate concentration, change in catalytic efficiency, change in the intracellular environment, and modification of the lipid membrane structure and function.

Supplement 5–1

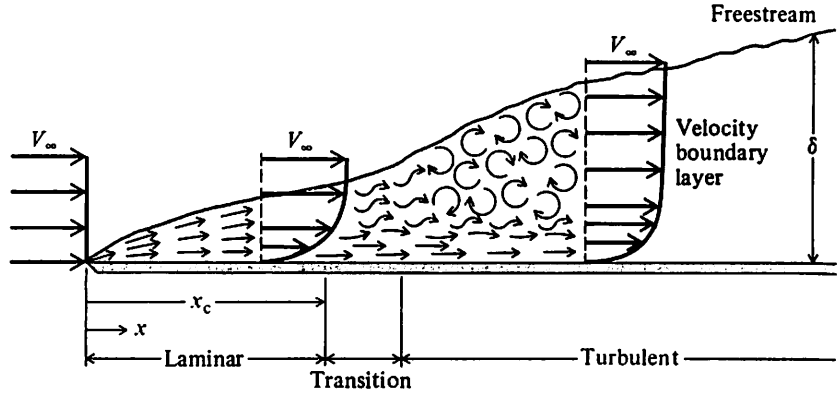
Convective Heat Transfer

Convection is the transfer of heat by movement of a fluid (either a liquid or a gas). Consider a flat plate of temperature T_{fp} that is immersed in a moving fluid with a free-stream temperature T_∞ (the ∞ doesn't mean the temperature is infinite, but is measured at an infinite distance from the plate). The fluid has a free-stream velocity (i.e., at infinite distance from the plate) of V_∞. Motion of the fluid will establish a boundary layer on the surface of the flat plate. The fluid is stationary at the immediate surface (zero velocity; this is the no-slip condition) and the velocity profile extends away from the plate until it equals the free-stream velocity. The boundary layer is defined as that region with velocity >0 and <0.99 V_∞. The thickness of the boundary layer (δ) increases with distance from the leading edge of the plate (x); it is thinnest at the leading edge and thickest at the trailing edge. The boundary layer thickness depends on many variables, including the free-stream velocity (V_∞), distance from the leading edge of the plate (x), and the fluid density (ρ) and viscosity (η).

The boundary layer is initially streamlined (laminar) but becomes turbulent at a critical distance (L_c) from the leading edge. The critical distance depends on the local Reynolds' number, a dimensionless coefficient defined as

$$R_e = V_\infty x_c/(\rho/\eta) = V_\infty x_c/\nu$$

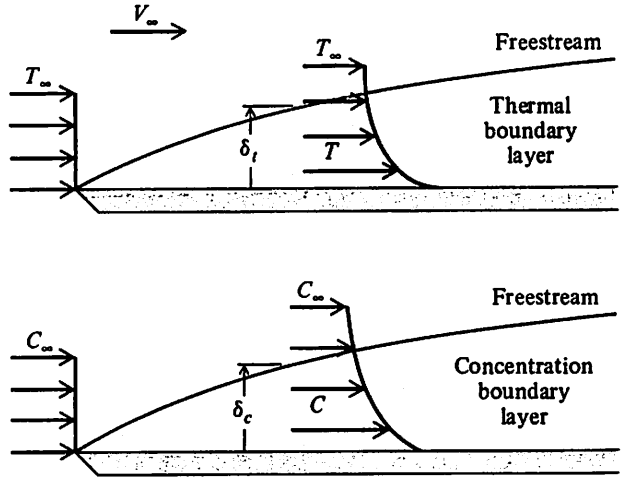
where ν is the kinematic viscosity ($1.5\ 10^{-5}$ for air and $1.0\ 10^{-6}$ $m^2\ sec^{-1}$ for water at 20° C). The Reynolds'



Velocity boundary layer.

number is essentially the ratio of inertial forces ($V_\infty x_c$) to viscous forces (ρ/η), and it indicates whether the flow is laminar (streamline flow, at low R_e) or turbulent (eddy flow, at high R_e). The critical R_e is generally about 5 10^5. The laminar boundary layer becomes turbulent at about 4.5 m from the leading edge and is about 0.04 m, for a flat plate in air (V_∞ = 1 m sec^{-1}).

There are not only velocity boundary layers around objects. For example, there is a thermal boundary layer around an object if there is a temperature difference between it and the fluid. There is a boundary layer of O_2-depleted water around aquatic animals. There is a relative humidity boundary layer around a moist-skinned animal in dry air. The same general considerations determine the thickness of these boundary layers as for velocity boundary layers, but it is important to appreciate that the thicknesses of the different types of boundary layers are not necessarily the same, or even of the same order of magnitude. (*See Incropera and Dewitt 1981.*)



Thermal and concentration boundary layers.

Supplement 5–2

Newtonian Model for Thermoregulation in Endothermic Mammals and Birds

The thermoregulatory strategy of endothermic mammals and birds is readily apparent from consideration of the effects of air temperature on the thermal balance of inanimate objects. The heat loss of an object is proportional to ($T_{obj} - T_a$), and therefore increases in a linear fashion at lowered T_a. The heat input needed to keep T_{obj} constant increases in a corresponding linear fashion with decreased T_a, i.e., heat input is proportional to ($T_{obj} - T_a$). A graph of heat loss as a function of ($T_{obj} - T_a$) for such a simple, Newtonian system would extrapolate to zero heat loss at $T_{obj} = T_a$. The slope of the relationship between heat input and T_a is thermal conductance (C);

$$\text{Heat Input} = C(T_{obj} - T_a)$$

This simple Newtonian model for heat balance of an inanimate object (with no evaporation of water) applies in principle to endothermic mammals and birds.

Let us consider a hypothetical endothermic mammal or bird that maintains a constant T_b over a wide range of T_a but has an elevated T_b when stressed at high T_a (facultative hyperthermia). The T_b is kept constant by the control of metabolic heat production. There is a similar hypothetical relationship between metabolic heat production (*MHP*) and body temperature (T_b) as a function of air temperature (T_a), for an endothermic mammal or bird, as for the inanimate object

$$MHP = C_{wet}(T_b - T_a)$$

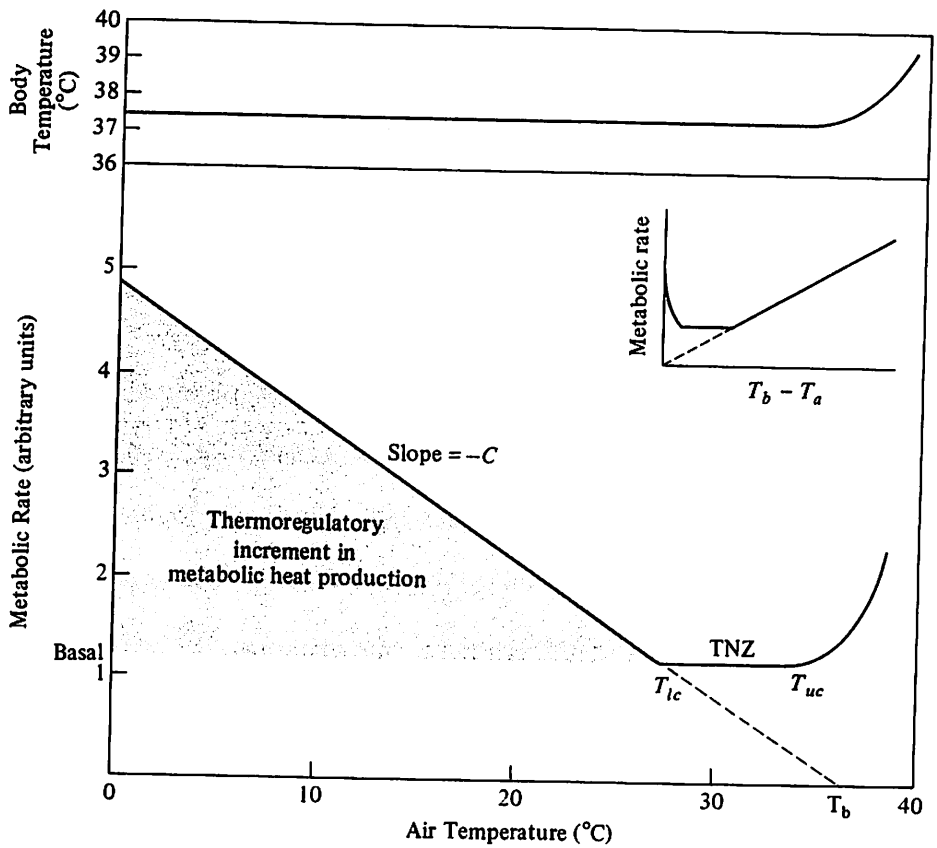
where C_{wet} is the wet thermal conductance (since animals invariably have some evaporative heat loss that contributes to heat dissipation). The MHP doesn't decline to 0 at $T_a = T_b$, but plateaus at a minimum value, the basal metabolic rate (*BMR*). The *BMR* is constant over a range of T_a, from the lower critical temperature (T_{lc}) to the upper critical temperature (T_{uc}). The relationship between MHP and $T_a < T_{lc}$ has a slope equal to −(thermal conductance) and extrapolates to T_b at MHP = 0. The figure inset shows the same relationship, but MHP is graphed as a function of $T_b - T_a$.

The wet thermal conductance is the thermal conductance uncorrected for evaporative heat loss; its units are ml O_2 g^{-1} hr^{-1} $°C^{-1}$ or J g^{-1} hr^{-1} $°C^{-1}$. The value of C_{wet} alters through the thermoneutral zone (being lowest at T_{lc} and highest at T_{uc}). This conductance change involves nonenergy requiring physiological changes, such as redistribution of blood flow to the skin, increased respiratory water loss, and behavioral responses (posture adjustment, pilo- or ptilo-depression of the fur/feathers). The VO_2 increases above T_{uc} because (1) T_b tends to increase, hence VO_2 is increased by a Q_{10} effect, and (2) there is a significant metabolic cost for many physiological responses to heat stress (panting, sweating, etc). The dry thermal conductance (C_{dry}) is the conductance corrected for evaporative heat loss (*EHL*; J g^{-1} h^{-1}).

$$(MHP - EHL) = C_{dry}(T_b - T_a)$$

Evaporative heat loss is calculated from the evaporative water loss (e.g., g H_2O g^{-1} hr^{-1}) and the latent heat of fusion (e.g., 2400 J g^{-1}).

Some endothermic mammals and birds conform to this hypothetical physical model for heat exchange but many



Effects of ambient temperature on metabolic rate and body temperature for a Newtonian model.

deviate somewhat from the model. For example, T_b often declines slightly at low T_a reflecting the gain of the thermoregulatory system. The slope of the relationship between *MHP* and $T_a < T_{lc}$ is not necessarily constant, and does not necessarily extrapolate to zero *MHP* at T_b. The dry thermal conductance can be markedly increased at high T_a, to facilitate passive heat dissipation.

Supplement 5–3

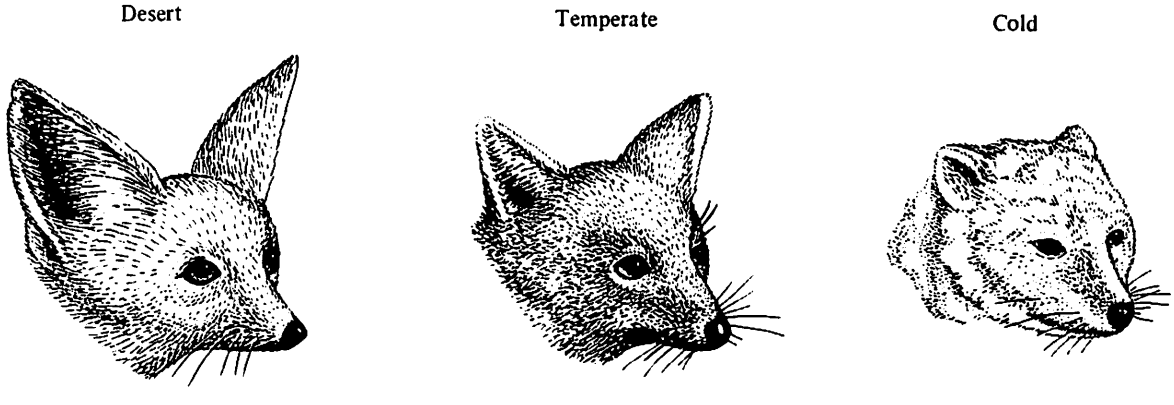
Bioclimatic Rules

Size, shape, and insulation are major determinants of heat exchange for endotherms. Consequently, it is logical to think that adaptation to cold climates might affect these aspects of an endotherm's morphology. A series of bioclimatic rules or laws have been proposed to explain adaptive climatic variation in body morphology. These rules are of interest because they usually reflect a mechanism for the adaptive modification of heat exchange, even though many may not be generally applicable but reflect specific adaptations in only certain animal taxa.

The bioclimatic laws were generally based on theory or circumstantial evidence, such as observed climatic trends in the morphology of certain species of endotherms, or of different geographic populations for a single species. Direct developmental evidence for bioclimatic rules has sometimes been obtained by raising endotherms (e.g., littermates) in differing T_a environments, and showing a direct ontogenetic effect of climate on body morphology.

In 1839, Sarrus and Rameaux postulated their surface rule: larger animals have a lower surface-to-volume ratio than do smaller animals. Consequently, larger endotherms would have a lower mass-specific heat loss and this would presumably be adaptive in a cold climate. Bergmann's rule makes the similar assertion that it is energetically less expensive for a large endotherm to survive in a cold climate because its mass-specific heat loss is lower, i.e., endotherms should be bigger in cold climates. There is evidence for Bergmann's rule in some taxa. For example, wood rats (*Neotoma*) are larger in colder climates, and also have a lower thermal conductance and a lower CT_{min}. The mean body mass of male humans increases by about 0.5 kg for every 1° C decline in mean annual temperature. However, similar climatic trends in body size are not observed for many species of endotherm. There is also no conclusive developmental evidence for Bergmann's rule in endotherms. Perhaps Bergmann's rule is not universal because many other cold-adaptations of endotherms can override body size effects. Another body mass rule, Cope's rule, relates body size to evolutionary history; the evolutionary trend within many taxa is towards larger size.

Allen's rule suggests that the heat loss is reduced for cold-adapted endotherms by a reduction in the size of their appendages, e.g., ears, digits, limbs. The lower surface-to-volume ratio of a smaller appendage reduces its heat loss. Circumstantial evidence for Allen's rule is again obtained from climatic trends in body morphology of related endothermic species. For example, arctic foxes have small ears compared to desert-adapted kit foxes; the desert fennec and bat-eared fox have extremely large ears, and temperate foxes have intermediate-sized ears. Arctic rabbits have smaller ears than desert jackrabbits.



Ear sizes for foxes from different climates.

Even cold-adapted races of humans tend to have shorter limbs and stockier bodies than desert-adapted races. Thomson's nose rule suggests that the human nose becomes relatively narrower at lower mean annual temperatures; the climatic significance of nose shape may be related to susceptibility to respiratory infections, or nasal countercurrent and water exchange. However, there may be reasons other than thermoregulation for appendage size; for example, bat-eared foxes have acute hearing for locating underground prey. There is developmental evidence for Allen's rule. Mice raised at low T_a have shorter tails than mice raised at high T_a. Pigs raised at low T_a have shorter tails, smaller ears, and stockier bodies than pigs raised at high T_a. A simple mechanism for these developmental changes is the effect of peripheral hypothermia on blood flow. Peripheral vasoconstriction would decrease nutrient delivery to the appendages and retard growth. For example, the fingernails of humans grow more slowly in cold climates.

Wilson's rule relates the thickness of insulation to climate. A thicker insulative layer is clearly adaptive to endotherms in cold climates. Arctic species have thicker coats than tropical species. There are also seasonal changes in the coat thickness for many arctic mammals; the coat is thinner in summer and thicker in winter. There is again developmental evidence for Wilson's rule. Pigs raised at low T_a have more hair than pigs raised at high T_a.

Gloger's rule suggests that animals have a lighter coat color in cold, wet climates and darker coats in warm, dry climates. Coat color clearly has many different roles, but it can affect thermal exchange. For example, the white fur of arctic mammals reflects solar radiation deep into the coat and facilitates thermoregulation. The white fur of polar bears may act as a light guide to facilitate deep penetration of light. (*See Ley 1971; McNab 1971; Hafez 1968; Damon 1975; Calder 1984; Kleiber 1975.*)

Recommended Reading

Alexandrov, V. Y. 1977. *Cells, molecules and temperature*. Berlin: Springer-Verlag.

Bakken, G. S. 1976. An improved method for determining thermal conductance and equilibrium body temperature with cooling curve experiments. *J. Thermal Biol.* 1:169–75.

Bakker, R. T. 1971. Dinosaur physiology and the origins of mammals. *Evolution* 25:636–58.

Bakker, R. T. 1972. Anatomical and ecological evidence of endothermy in dinosaurs. *Nature* 238:81–5.

Bartholomew, G. A. 1981. Physiological thermoregulation. In *Biology of the reptilia*. Vol. 12, *Physiological ecology*, edited by C. Gans and F. H. Pough, 167–212. New York: Academic Press.

Benton, M. J. 1979. Ectothermy and the success of dinosaurs. *Evolution* 33:983–97.

Calder, W. A., and J. R. King. 1974. Thermal and caloric relations of birds. In *Avian biology*, edited by D. S. Farner and J. R. King, Vol. IV, 259–413. New York: Academic Press.

Carey, F. G. 1982. A brain heater in swordfish. *Science* 216:1327–29.

Casey, T. M. 1981. Behavioral mechanisms of thermoregulation. In *Insect thermoregulation*, edited by B. Heinrich, 80–114. New York: John Wiley & Sons.

Cossins, A. R., and K. Bowler. 1987. *Temperature biology of animals*. London: Chapman & Hall.

Hardy, R. N. 1978. *Temperature in animal life*. Baltimore: Univ. Park Press.

Hazel, J. R., and C. L. Prosser. 1974. Molecular mechanisms of temperature compensation in poikilotherms. *Physiol. Rev.* 54:620–77.

Heinrich, B. 1981. *Insect thermoregulation*. New York: John Wiley & Sons.

Hew, C. L., G. K. Scott, and P. L. Davies. 1986. Molecular biology of antifreeze. In *Living in the cold: Physiological and biochemical adaptations*, edited by H. C. Heller, X. J. Musacchia, and L. C. H. Wang, 117–23. New York: Elsevier.

Hochachka, P. W., and G. N. Somero. 1984. *Biochemical adaptation*. Princeton: Princeton University Press.

Huey, R. B. 1982. Temperature, physiology, and the ecology of reptiles. In *Biology of the reptilia*. Vol. 12, *Physiological ecology*, edited by F. H. Pough, 26–90. London: Academic Press.

McGowan, C. 1979. Selection pressure for high body temperatures: Implications for dinosaurs. *Paleobiology* 5:285–95.

Monteith, J. L. 1973. *Principles of environmental physics*. London: Edward Arnold.

Pough, F. H. 1980. The advantages of ectothermy for tetrapods. *Am. Nat.* 115:92–112.

Precht, H., J. Christophersen, H. Hensel, and W. Larcher. 1973. *Temperature and life*. Berlin: Springer-Verlag.

Stevens, E. D., and W. H. Neill. 1978. Body temperature relations of tuna, especially skipjack. In *Fish Physiology*. Vol. VII, *Locomotion*, edited by W. S. Hoar and D. J. Randall, 315–424. New York: Academic Press.

Thomas, R. D. K., and E. C. Olson. 1980. *A cold look at the warm-blooded dinosaurs*, AAAS Selected Symp 28. Washington: AAAS.